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THE
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VOL. XXXII ST. LOUIS, JANUARY, 1922 No. 1

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE OPERATIVE TREATMENT OF SUPPURATIVE MENINGITIS WITH ESPECIAL REFERENCE TO IRRIGATION OF THE CRANIAL AND SPINAL SUBARACHNOID SPACES; AND THE IMPORTANCE OF PROTECTIVE MENINGITIS FROM A PROGNOSTIC AND THERAPEUTIC STANDPOINT, WITH AN ANALYSIS OF THE CASES OF RECOVERY—EXCLUSIVE OF MENINGOCOCCIC—REPORTED IN THE LITERATURE.*

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INTRODUCTION.

This paper is presented at length because a compilation by the author, assisted by Dr. F. Robbins, of all the recorded cases of recovery from suppurative meningitis—exclusive of meningococcus mengingitis—shows that the number of true cases is even smaller than is generally supposed. Many of the reports in which an operation is said to have been followed by recovery contain no positive evidence—the evidence, of micro-organisms in the fluid obtained by lumbar puncture—of a bacterial invasion of the general cerebro-spinal fluid system, having been present. In fact many reports frankly state that the fluid was sterile. Doubtless the majority—if not all—of these cases were suffering, not from a suppurative but from a protective meningitis, the reporters evidently regarding these essentially different processes as identical. This paper is given with the hope that by a re-study of the patological, clinical

*President's Address. Read before the American Otological Society at its fifty-fourth annual meeting, Atlantic City, June 1, 1921 (read in part).

and experimental evidence now available the surgeon of the future may come, not only to a better understanding of this group of diseases, but to a more rational and successful treatment of them.

In the light of recent experiments in induced suppurative meningitis we should revise some of the opinions previously accepted. In meningitis we are dealing with a septic process confined to a physiological system of the brain—a circulatory system that is anatomically as distinct and definite as the blood or the lymphatic systems.

Infection of this circulatory system—suppurative meningitis—causes profound toxemia, because, with an inflammatory process within the cerebro-spinal fluid system, the products of the inflammation, its toxins, etc., are poured into the general blood circulatory system, into which it empties by osmosis.

In suppurative meningitis the brain tissue is only secondarily involved, and consequently cerebral compression, which plays such a large and important role in *cerebral tissue suppuration*—or brain abscess—is but seldom present. When present it is secondary to obstruction in the cerebro-spinal fluid system and not to increase in the brain bulk, as in brain abscess.

The fundamental facts that (1), suppurative meningitis is primarily a disease of the cerebro-spinal fluid system—a circulatory system which, while intimately associated with the cerebral tissue and blood circulatory system, is still distinct from it—and that (2), the cerebral tissue is only later and secondarily involved; are of prime importance in approaching the subject from a surgical standpoint. Consequently in thinking of suppurative meningitis we should not regard it as in any way analogous to brain abscess, but rather as akin to a combination of thrombosis and a cerebral arterial embolism—the former a septic process and the latter causing a disturbance in the nutrition and metabolism of the cerebral tissue. Even this analogy is so imperfect that there is no real profit to be derived from its application.

CLASSIFICATION.

Suppurative Meningitis a Group of Diseases.

Clinically as well as experimentally suppurative meningitis should not be regarded as a disease but as a group of diseases of the cerebro-spinal fluid circulatory system. From an etiological and general standpoint they may be divided as follows:[1]

1. Meningitis from a localized extra-dural focus of adjacent infection—ear or accessory sinus—the meningitis occurring either

through direct extension, thrombophlebitis, or "from trauma, with secondary general spread." (Netter.)

2. Meningitis secondary to focal brain abscess (Bull, 1916; Detweiler and Maitland.)

3. Meningitis as part of an overwhelming blood infection, in which meningitis is only one, and frequently the least conspicuous lesion. (Bull, 1917.)

4. Meningitis generalized from the beginning, which appears to be the cause of death rather than the septicemia or its accompanying lesions. (Austrian, Elser and Huntoon.)

While the etiological process must always be the commanding factor in deciding whether or not a disease is amenable to surgery—for to operate for a local manifestation of a general disease must necessarily, in the vast majority of cases be fruitless—the actual anatomico—pathological condition presents becomes of prime importance once it has been decided to attack a supperative meningitis, surgically.

Surgically, then, cases of suppurative meningitis should be divided into

(1)—fulminating types;

(2)—exudative types,

(a) acute exudative,

(b) subacute and chronic exudative;

(3)—protective types.

The surgical procedure to be adopted in an individual case, if any degree of success is to be obtained, must take into consideration both the etiological factor and the pathological condition present.

ANATOMY OF THE CEREBRO-SPINAL FLUID SYSTEM.

The cerebro-spinal fluid circulates within closed cavities and tubes —the ventricles, the cisterna and the sub-arachnoid spaces constituting the cerebro-spinal circulatory system—somewhat as the blood circulates in the blood vascular system; and like the walls of the vascular system, the walls of the cerebro-spinal circulatory system are normally impermeable to the contained fluid.

The cerebro-spinal fluid finally empties into the blood stream after performing its functions in the brain, passing by osmosis into the venous sinuses of the head, and at certain points, where the system has passed outside of the cranium, communicates again by osmosis, with the extra-cranial lymphatic system. This communication occurs along the arachnoid prolongations around the sheaths of certain nerves, especially the olfactory and the auditory.

Thus while the cerebro-spinal fluid circulatory system is a closed system without an apparatus for the punification and return of its fluid, such as the vascular system has in the heart and lungs, it utilizes both the vascular and lymphatic systems for its discarded, material, much in the same way as the blood vascular system utilizes the kidneys; with this difference, however, that while the blood discharges through the kidneys only its deleterious material, the entire cerebro-spinal fluid constantly is being discharged into the blood or the lymphatic system.

Viewed, then in a large way, both anatomically and physiologically, the cerebro-spinal fluid circulatory system *must* be of importance to the cerebral functions. Why its study was so long disregarded is to be explained by the delicacy of its anatomy.[2 11]

The circulating cerebro-spinal fluid is produced chiefly from the choroid plexus within the ventricles, the walls of which are lined by cells, the ependyma, which are impervious to the fluid. From the ventricles the fluid passes by a process of overflow through the foramen of Sylvius into the fourth ventricle, leaving it by the medial foramen of Magendie and the lateral foramina of Luschka; from these it spreads over the base of the brain, and accumulates in the large basal cisterna, whence it is distributed, by way of the sponge-like communicating meshes of the sub-arachnoid spaces over the cortex, along the vessels and convolutions, emptying at last, by osmosis, into the venous sinuses.

In its passage over the cortex there is contributed from the cerebral substance—through the prolongations of the sub-arachnoid spaces along the vessels entering or leaving the cerebral tissue called the perivascular spaces—a small amount of crebro-spinal fluid which has a somewhat different composition from the cerebro-spinal fluid formed in the ventricles. These perivascular channels, opening directly into the sub-arachnoid spaces of the cortex, are undoubtedly the means of direct communication between the intra-cellular juices of the brain cells—the perineural system—and the general cerebro-spinal circulatory system.

The cerebro-spinal fluid acts to remove the waste products of the brain, but whether it supplies any nutritive ingredient necessary to the continued functioning of the brain is not known. It is inconceivable that it does not possess some such quality, especially as one of its normal contents is a sugar reacting substance.

Impregnability of Cerebro-Spinal Fluid System: The cerebro-spinal fluid system, while anatomically composed of very delicate cells, is very resistant to infection when attacked either upon its

external surface, or internally, from the cerebral tissue; and while in close anatomical proximity to the blood circulatory system—being separated from the pial vessels by only one layer of cells and actually surrounding the vessels as perivascular spaces—when uninjured the cerebro-spinal fluid system does not allow of the invasion of the sub-arachnoid spaces by bacteria circulating free in the blood stream. The cells of the arachnoid offer an efficient barrier to such an invasion. With micro-organisms circulating free in the blood stream, however, a slight disturbance of the normal protective mechanism—such as would be occasioned by the withdrawal of a small amount of the cerebro-spinal fluid; the presence of an asceptic meningitis; or the temporary stoppage of the blood current—is enough to permit the passage of the free bacteria from the blood system into the cerebro-spinal fluid system, and a suppurative meningitis follows.

Bacteriological Classification of Meningitis: Clinically, otologists have come to recognize three different types of meningitis, the differentiation largly being based upon (a), the supposedly different degrees of virulence of the infecting micro-organisms in the leptomeninges, with (b), the associated alterations in the cerebro-spinal fluid, (c), the proportionate rapidity of a fatal termination, and (d), the different post mortem findings.

The classification at present generally accepted is—

I—*Fulminating Pneumococcus Leptomeningitis,* (frequently described as streptococcus mucosa). This type of meningitis usually is regarded as coincident with a general blood-stream infection, although personally I have never accepted this view. In my experience nearly all of the cases have been preceded by an acute otitis media or a mastoid involvement. These cases all ended fatally, sometimes very quickly, and the post mortems revealed a general leptomeningitis with numerous hemorrhages into the piarachnoid, with or without a small amount of exudate.

II—*Streptococcus and Staphylococcus Meningitis,* (much more common, but not so virulent), which generally follows a chronic or acute local focus of adjacent infection—otitis, sinusitis, or cranial osteitis such as accompanies an infected compound fracture of the skull—and which surgeons have come to regard as the breaking down of an initial local immunity, generally through direct extension into the meninges. (Koerner). These cases while of much longer duration than the former, have an almost equal fatality, the post mortem invariably showing great areas of exudation covering the basal cisterna.[12]

And, least virulent of all;

III—*Meningococcus Meningitis,* with a cloudy or opaque cerebro-spinal fluid, containing, however, comparatively few micro-organisms. This type, even before the discovery of a specific serum had a fair proportion of recoveries.

Clinical Classification:

Fulminating pneumococcus meningitis, of acute aural, accessory sinus or blood-stream origin; streptococcus meningitis, of chronic or acute aural, nasal or cranial bone origin: and meningococcus meningitis, probably of nasal or blood-stream origin, are clinical entities. Fulminating pneumococcus meningitis is characterized by a high virulence of the micro-organisms, and imperfect or absent tissue protective reaction. The mode of invasion is unknown, but probably is from a direct invasion of the sub-arachnoid spaces through the blood vessels, or of the sub-arachnoid space prolongations along the olfactory nerve. Meningococcus meningitis is characterized by a prompt tissue protective reaction as shown by the early appearance of a cloudy fluid filled with leucocytes, with but few micro-organisms, the latter disappearing either spontaneously or after the injection of serum or antiseptics. The mode of invasion in this type also is unknown, but probably is directly from the nasal mucous membrane, or from the blood-stream itself, as a majority of the cases are found to have a blood-stream infection concurrent with the meningitis. Midway between these two types, both in virulence and in duration, stands the streptococcus and staphylococcus meningitis of local origin.

Effect of Classification on Treatment:

With the acceptance of such a classification it was natural that treatment should be largely empirical and based upon attempts at (1), the establishment of drainage, (2), the injection of a specific serum, and (3), the relief of compression. Belief in a concomitant blood-stream infection in pneumococcus meningitis caused the surgeon to throw up his hands as soon as pneumococci were demonstrated in the cerebro-spinal fluid.

It is one of the purposes of this paper to emphasize my belief that, if progress is to be made in the surgical treatment of meningitis, this generally accepted bacteriological classification must, to a large extent at least, be superseded by a classification which is based upon the etiological factors and the pathological findings in experimentally produce meningitis; for it is by experiment alone that the location, course, and extent of the lesions during the initial period can be discovered.

Experimental Meningitis:

The knowledge derived from the experimental production of purulent meningitis—chiefly by Weed, Wegeforth, Ayer, Felton and Essick[13—16] should, I think, cause us greatly to modify our views, as it makes possible a more philosophical understanding of meningitis as a whole, and, consequently, a more rational line of treatment. To accomplish this, however, I believe it is necessary that we make an effort to visualize the pathological process in its different stages, *modibed as it is by the protective and reactive influences of the pro tective meningeal mechanism.*

Experimental evidence favors the view that the grade of the meningitis depends, not entirely upon the *kind* of invading micro-organisms, but also upon the quantity of them, as all types of pyogenic micro-oragnisms, if implanted in sufficient quantity in the sub-arachnoid spaces, will induce a meningitis of greater or less severity.[17]

This being so, then in many cases there must be a period of latency when the micro-organisms, although virulent, are not of sufficient number to obtain command. Such is indeed clinically the case in the latent period of pneumococcic infection, as recorded by many observers; when, with pneumococcic type III organisms in the cerebro-spinal fluid, there are few or no clinical manifestations of meningitis.

Experimentally produced meningitis from a blood-stream infection, demonstrates that in meningitis of blood-stream origin at least, the meningitis (1), originates in the sub-arachnoid spaces chiefly *over the cortex* of the cerebral hemispheres; and further that (2), the infection is at first confined to the sub-arachnoid spaces of the cerebro-spinal system, and (3), extends in a direction contrariwise to the direction of the cerebro-spinal fluid circulation, namely, from the cortex to the basal cysterna; that (4), as long as the infection is thus confined there is no *involvement of the cerebral tissue itself,* whatever changes occur in the tissue being of a protective and not of an infective nature; but (5), on *reaching the ventricles* infection of the brain tissue appears, and also infection of the blood vessels of this region; that (6), the outstanding pathological manifestation of septic infection of the cerebro-spinal fluid system is the production of an exudate, in part inflammatory and in part protective, which plugs the cerebro-spinal circulatory system, *and so prevents proper cerebral functioning.*

TOXICOLOGY OF THE CEREBRO-SPINAL FLUID SYSTEM.

Effect of Alterations in the Chemical Composition and Temperature Changes in Fluids Passed Through the System: Weed and

Wegeforth[18] demonstrated that apparently trivial alterations in the chemical composition of the fluid *passing hrough* the cerebro-spinal fluid system are highly toxic; even if neutral in composition, the fluid must contain a small proportion of calcium and the temperature of the fluid must be at about that of the body; otherwise lethal, respiratory and circulatory disturbances associated with acute nausea quickly follow.

Foreign substances *injected into* the sub-arachnoid space, on the other hand, while causing marked symptoms of general body reaction,are not necessarily followed by death.[19]

SURGICAL TYPES OF MENINGITIS.

Adaptations of Clinical Types to Pathological Lesions Occurring in Experimentally Produced Meningitis: The clinical manifestations and post-mortem findings in cases of suppurative meningitis being similar to the clinical history and pathological lesions of experimentally produced meningitis, I believe that the generally accepted bacteriological classification should largely be abandoned and for surgical purposes the etiologico-pathological classification of produced meningitis (Ayers) adopted; suppurative meningitis being, as already stated, divided into four types.

In the first type—or the Acute Fulminating Type—the outspoken symptoms are, a profound blood-stream sepsis, rapid pulse, high temperature, great prostration, plus the presence of a *distinctly cerebral* element manifested by intense headache, vomiting, restlessness, delirium, and a death clinically different from the death by exhaustion usual in sepsis. The fatal termination often occurs very suddenly while the patient apparently is in good health. This form of death has been explained as due to a "derangement of the controlling cerebral mechanism."[20]

Post-mortem examination shows multiple hemorrhages in the pia-arachnoid—an evidence of the virulence of the poisoning in the blood vessels. This type corresponds in all particulars to the acute fulminating cases of pneumococcus meningitis. This fulminating type, however, frequently is preceded by periods of quiescence in which, although the micro-organisms are present in the cerebro-spinal fluid, few symptoms are manifest; the patient apparently being fairly well, the only clinical evidence of the impending fulminating meningitis being a moderate and irregular elevation of temperature, possibly irregular vomiting and headache—all of them symptoms of cerebral suppuration.

General Blood-Stream Infection Secondary to the Fulminating Meningitis:

In the fulminating meningitis of otitic origin, at least, I believe the general blood-stream infection is secondary to the meningitis, as an otitis invariably *precedes* the meningitis for varying periods, although in a few cases in my expreience a blood-stream infection apparently was present from the beginning. In one case death occurred 72 hours after the initial earache, the post-mortem showing hemorrhages in the pia, while the serous fluid within the nasal accessory sinuses was loaded with the invading micro-organisms. To both of these pathological conditions I was the first, so far as I am aware, to call attention.[21]

Although some of my cases of sudden fulminating meningitis following a mastoid operation were doubtless latent meningeal infections from an early period of illness, as a careful examination of the history of Case VIII will demonstrate; in all of them nevertheless, an aural suppuration preceded the meningeal manifestations by an appreciable interval. (See also Case V).

Examination of the cerebro-spinal fluid at this time may show but moderately increased pressure, a slightly increased cell count and an apparent absence of micro-organisms, the latter being so few as to be overlooked for considerable periods of time, even by expert bacteriologists. Combined with the fact that at the outbreak of the meningeal manifestations the invading micro-organisms in the cerebro-spinal fluid were so few as on numerous occasions to be overlooked, I am persuaded that these cases originally are a strictly local aural, or possibly nasal, infection, the succeeding meningitis being of such severity as to occasion more or less promptly a blood-stream infection.

This view is substantiated by the clinical observation of the invariable absence of micro-organisms in the blood culture in the early stages of the disease, and by the experimental observations of Felton and Wegeforth (loc. cit.): "In all acute fatal cases of experimentally induced meningitis caused by the injection of micro-organisms into the sub-arachnoid space, every culture of the heart's blood showed the presence of the same bacteria with which the animal had been inoculated intra-spinously."

And more conclusively still if the animal recovered from the meningitis and later died, "the micro-organisms could still be obtained from the blood, although the meninges were clear."

From clinical and experimental evidence, then, fulminating meningitis (pneumococcus), when preceded by a known focus of primary infection (in the ear or nasal sinuses), should be regarded as a *secondary* infection, the meningitis itself—by an overwhelming attack of virulent micro-organisms—causing an almost immoderate blood-stream infection by their direct action on the blood vessels. Those cases in which an apparently sudden onset and a rapid course

are followed by a fatal termination, after a period of quiescence, would suggest that at a certain point the protective forces within the meninges, which have been in control, are suddenly overpowered; thus allowing complete bacterial domination—especially as the sudden outburst at times is associated with the clinical picture of profound hemogenic toxemia, as well as of cerebral function involvement. Such a view, although precluding surgical intervention in the acutely fulminating cases offers a much more favorable outlook in the cases in which the blood vessels are not immediately attacked than if we regard the meningitis as concurrent with a general blood-stream infection.

Case I. Acute Fulmination (Pneumococcic Type III) Meningitis:

Synopsis: Following an aural suppuration, outspoken meningeal manifestations on the third day; rapid death. *Post Mortem*: Pneumococci in sub-arachnoid spaces; slight exudate; numerous hemorrhages in pia, protective leucocytic infiltration of cerebral cortex; pnumococcic invasion of affected mastoid and of the mucous membrane of several nasal accessory sinuses; the latter being possibly a secondary infection from the blood-stream, although a negative blood culture was obtained but a few hours before death, and there was no evidence of protective reaction in the mucous membrane of the accessory sinuses, as would have been the case if the nasal sinuses were the primary focus of infection.

Clinical History: On January 20, 1918, patient had his first ear ache. When he arrived at hospital on January 21st, the ear was discharging a waterish, yellow exudate. This continued on the 22nd and 23rd. Examined repeatedly by Dr. Earl Brooks, who said he might have had a little tenderness over the mastoid, but thought not. Patient had been feeling comparatively well, so far as his neighbors in the ward knew. His temperature, however, on the 23rd reached 102°; on the 24th, 101⅘°; late in the afternoon of the 24th, 102⅘°. The nurse stated that during the whole of the 24th the patient seemed stuporous, but was easily aroused and talked. At 3:30 he vomited while having his nose sprayed. At 5 o'clock he sat up in bed and said to the nurse, "I do not think I can hold the basin to have my ear syringed," but he did hold it. About 5:15 he asked the nurse to give him a drink of water. I had passed the patient's bed at 4 o'clock and had not noticed his appearing sick; but suddenly at 6 o'clock, while I was in the ward, the man vomited and had a convulsion, and when I reached his bed he was in coma, with widely dilated pupils and nystagmoid movements of his eyes.

Following this attack the patient became slightly conscious, but could not be fully aroused. Lumbar puncture at 7 p. m.; the fluid, under great pressure, was rather cloudy, with grey streaks—looked like the fluid seen in epidemic cerebro-spinal meningitis, and I thought this probably was the case. Examination of the eyes was negative. The bacteriological examination showed pneumococci. The precipitin test was positive for type III. A blood culture was negative. About 8 o'clock he had a rather active chill and his temperature rose rapidly. Death in deep coma, within 6 hours of the initial convulsion.

Autopsy and Report by Dr. B. F. Weems, jr.

Clinical Diagnosis: Pneumococcus Meningitis.

Anatomical Diagnosis: Otitis media and mastoiditis (left side); ethmoiditis and right frontal sinusitis; cerebro-spinal meningitis.

External Examination: Body of adult white male, approximately 23 years of age, medium size, well developed and nourished, rather

muscular. Adenoid type of facies, poorly developed dental arches, and weak chin. Bodily conformation and hair distribution distinctly masculine. Rigor mortis general and very pronounced; the usual purple blotches of hypostatic congestion seen on dependent portions of skin. External genitalia normal. Orifices negative.

Internal Examination: *Brain*: Calvarium was removed in the usual manner and the brain lifted out without separating the dura from the hemispheres. An abnormal cloudiness was apparent in the piarachnoid spaces at the base of the brain. There was a general engorgement of the vessels of the entire brain, those of the cerebral cortex especially. The usual cloudiness due to coagulated lymph along the larger vessels in the Sylvian fissure and some of the sulci, was much augmented by a cellular exudate. There was no appearance of a frankly purulent exudate, however. *The most marked characteristic of the picture was due to the innumerable capillary hemorrhages scattered over almost the entire surface.* These appeared to be just beneath the pia, the extravasated blood spreading out in a thin film over the brain surface. The film was thin enough and extensive enough over the cerebellum to give the whole of the posterior and inferior surface of this organ an almost homogeneous pink color. The hemorrhagic condition was *most in evidence over both temporal lobes.* Here it was apparent that many little hemorrhages had taken place from the small arterioles that entered the cortex. Upon the anterior and superior curvatures the hemorrhagic process was less developed.

Base of Skull: Nothing abnormal was to be observed in the dura or about the nerve endings and foramina at the base of the brain. The antrum and the neighboring cells in the petrous portion of the temporal bone were separated from the overlying dura by a thin, almost transparent membrane. *This thin membranous character of the bony wall was noticed only in several small spots;* otherwise the surface appeared perfectly normal and unbroken. Within the mastoid antrum and the adjoining mastoid cells there was a slightly turbid exudate. The mucous membrane was everywhere thickened, soft and turgescent. *Deeper down in the middle ear there seemed to be little or no exudate.* The antrum and mastoid cells in the right temporal bone were found to be perfectly normal.

The ethmoid cells displayed the same juicy condition, all the cells being filled with slightly turbid fluid, and *the mucous membrane being much swollen and soft.* On the right side the sphenoid sinus contained some thin mucous secretion and the membrane appeared swollen. The cells of the right frontal sinus appeared very much like those of the left mastoid. They were filled with thin, turbid exudate and the mucous membrane was even more striking in its thickened, turbid character. Several very small capillary hemorrhages were to be seen in its substance.

Anterior mediastinum appeared normal. *Thymus gland* was quite large, about 9 cm. long by, perhaps, 5 cm. wide, and one cm. thick. It was removed in toto and preserved for closer study. Both pleural spaces were perfectly normal.

Lungs: Both organs presented a typical picture of acute congestion. The consistency was rather firm, but homogeneous; the cut surface very moist, smooth and of a dark red color. Considerable bloody fluid could be scraped up on the blade of the knife. Some air was contained in all parts of the tissue folds. Bronchi and vessels appeared quite normal.

Heart: Pericardium normal. Beneath the epicardium of the left ventricle there were numerous petechial hemorrhages. This ventricle was in tight contraction, while the right side of the heart was quite flaccid. Endocardium, valves and heart muscle normal.

Peritoneum: Smooth, moist and glistening. Appendix deep in the iliac fossa. Transverse colon hung down below the umbilicus. Mesenteric glands appeared quite normal.

Spleen: Malpighian follicles stood out very prominently, otherwise organ was normal.

Kidneys: A very perfect example of the *Horseshoe Kidney. The pelves* and the two separate ureters being perfectly developed. The pelves were situated anteriorly to the kidney substance and the urethers passed downward over the bend of the horseshoe. The capsule stripped readily at the left pole. There was no evidnce of abnormal condition on the surface. The organ was not sectioned.

Liver: Showed some acute congestion.

Pancreas: Appeared likewise to be somewhat congested.

Gall-Bladder: Distended with normal looking bile; no stones. No further dissection was made.

BACTERIO-HISTOLOGICAL REPORT.

a. Smear of fluid at base of brain showed polymorphonuclear leucocytes and abundant pneumococci. Culture gave pneumococci in pure growth.

b. Brain hardened in 4% formaldehyde and sections showing lateral surface of right hemisphere and median surface of left were prepared for museum.

c. Sections of cortex in temporal region showed a mixed hemorrhagic and purulent exudate in the spaces of the piarachnoid, in which a few pairs of pneumococci could be demonstrated. Many polymorphonuclear leucocytes could be seen passing through the walls of the capillaries in the superficial layers and invading the cortex.

N. B. The cortex, while the seat of polymorphic leucocytes—a protective reaction—was not invaded by the micro-organism itself. (W.P.E.)

d. Dura, over left temporal bone, was extensively invaded with polymorphonuclears, indicating the penetrating infection.

e. Smear from mastoid cells showed an occasional pneumococcus, many polynuclears. Culture showed pneumococcus.

f. Smear from the middle ear showed pus cells but no cocci could be found. Culture gave pneumococci.

g. Smear from ethmoid cells showed many pneumococci. Culture gave pneumococci and staphylococcuc auress.

h. Smears from mucous of right sphenoid sinus showed innumerable Gram negative and positive cocci and bacilli. Culture showed similar mixed flora.

i. Smears from mucous membrane of frontal sinus showed pneumococci in abundance. Culture gave pneumococci.

j. Sections of the tissue of the frontal sinus showed a *very marked edema of the sub-mucous tissues, especially of the epithelium and the adjacent layer.* Pneumococci found in sections.

k. Thymus was rather larger than usual.

l. Sections of the lung showed the typical picture of acute congestion and edema.

m. Horseshoe kidney preserved as a museum specimen.

n. Liver sections showed *engorgement of the capillaries about the intra-lobular vein,* with slight vacuolization of the liver cells in this region. Also there was a slight *general infiltration of the liver tissue with polymorphonuclear leucocytes. No abscess formation, but more leucocytes wandering out of the blood vessels than is normally the case.*

In the majority of cases, however, (even those of pneumococcic type III, infection), the bacterial invasion is not so overpowering —the bacteria being either too few, or else limited by the protective meningeal mechanism: in which case we have an exudative type—a much longer process, clinically and pathologically different.

II. *The Exudative Types*—the usual form of suppurative meningitis from direct extension of a streptococci or staphylococcic in-

fection of an adjacent structure, as the ear, nasal sinus, or bony cranium.

II—(a) *Acute Exudative*: The exudative type in reality is but a milder degree of the first, or fulminating type. *It has this difference, however, that part of the pathological process is not bacterial, but protective,* as the exudate is partly the product of the protective choreo-meningeal mechanism in its efforts to limit the bacterial and toxic influences. This exudate, however, by its presence adds to the damage by interfering with the cerebro-spinal fluid circulation, unless it is limited to a small area, and thus, a vicious circle is established to the advantage of increased bacterial growth and extension. It is this exudate which, by filling the sub-arachnoid spaces, and thus disturbing the proper functioning of the brain, causes the earlier cerebral manifestations; the accompanying symptoms of toxemia originate from the products of the inflammation discharged into the blood-stream by the cerebro-spinal fluid; and the terminal symptoms of cerebral compression are due to the invasion of the foramina of Luschka and Magendie[22 25] from extension of the exudate into the ventricles and the cerebral tissue.

The patient usually lives from ten to fourteen days, or even longer, during which time there may be periods of apparent recession of the infection, giving hope of ultimate recovery, only to the end, however, in all but very exceptional cases in death.

Case II; EXUDATIVE MENINGITIS (of pneumococcic origin);

Child, seven years of age. Seen in consultation with Dr. B. B. Ranson and Dr. Linn Emerson.

Synopsis: Aural suppuration, followed by a quiescent period, with acute meningeal onset, micro-organisms early present, but in such small numbers as to be overlooked by one competent pathologist. Periods of recession.

History: Two years previously had discharge from ears off and on, but otherwise well. Following an attack of measles, had adenoids and tonsils removed. Following tonsil operation, child again developed a running ear: apparently doing well for about two weeks, when, on the night of February 23, she suddenly complained of severe pain in the head and vomited. Temperature rapidly rose to 104°. Lumbar puncture; fluid clear, under pressure. The pathologist, Dr. B. F. Cline, reported that penumococci were present in the cerebro-spinal fluid, he having located them after a careful search of two hours.

With this history, there being no tenderness over the mastoid, a pneumococcus meningitis was diagnosed. The child was taken to the hospital for sub-arachnoid irrigation. Upon arrival at the hospital another lumbar puncture was performed. The hospital pathologist reported that there were no micro-organisms present, and that in his opinion it was a protective type of meningitis. The child had now been sick less than twenty-four hours. Her temperature was high, she had a rapid pulse, was complaining of severe headache and was vomiting, but she did not appear very sick. Blood culture was negative; leucocytes, 19,000; hemiglobin, 60-70 per cent.

Operation: Mastoid freely opened and nothing of importance found. Two trephined holes were drilled in frontal region, but on receiving the pathologist's report it was decided not to irrigate, especially as the child appeared to be in excellent condition.

Following the operation the child appeared better, but the temperature continued high. That night, for the first time, she was slightly delirious.

February 24 (the following day): Child vomited several times. Complained of some headache, but passed a very restful day and on the whole looked much better, although temperature still continued high and pulse fast.

February 26: Passed a restless, day, sleeping at intervals. During the day child again vomited several times. Lumbar puncture performed; this beoing the third lumbar puncture; for the first time the fluid was found to be cloudy under slight pressure; globulin increased; Fehling's solution reduced normally. The smears showed the presence of Gram positive diplococci. Cell count 2766 per cm. Cultures of fluid demonstrated pneumococci.

February 27: Was slightly delirious at times. Child, however, looked well and parents decided to remove it to its home, where it continued for two weeks, at times better and again worse, and at last died as the result of the meningitis.

Comment: A mistake was made in not acting on the examination of Dr. Cline, who reported finding pneumococci after two hours' search. For at this time the process undoubtedly was very early, as shown by the clear fluid and the low cell count with but few micro-organisms. Also, the history was so positive that we predicted a fatal termination. With such data, we should have washed out the sub-arachnoid space. The fact that the child lived for two weeks after the onset of the meningitis shows that it might have been possible to control the infection at this time. This child really died, not so much of the meningitis as of the associated septicemia.

II—(b)—*Sub-Acute, or Chronic Exudative Type*[26]—a much less common type, in which the bacteria, having invaded the sub-arachnoid spaces, are temporarily or permanently overcome by the protective meningeal mechanism. Experimental evidence has demonstrated, however, that the invading bacteria may take refuge in the arachnoid recesses around the nerves and blood vessels, from which lodging place they are capable of causing slow death by blood-stream infection, or of suddenly producing an acute exudative or fulminating meningitis.

Case III. Fulminating Meningitis from Recrudesence by Exposure and Vaccine Treatment of a Previous Meningeal Infection.

Male Adult: The *previous history* revealed in 1915 an infection of the ear followed by coma, with a sterile cerebro-spinal fluid. A left-sided mastoid operation was performed and the dura incised. Following this operation the cerebral symptoms disappeared to a large extent, but six weeks later an extra-dural abscess was located over the cerebellum. This was evacuated and the patient made an uneventful recovery, the wound healed and he had no further symptoms for two years.

Present Illness:

He complained of general malaise following his last injection of triple typhoid vaccine on Oct. 18, 1917. On Oct. 20 he had a full feeling in the left ear. On Oct. 21 he visited Dr. Blair Sutphen, who removed a small quantity of cerumen from the left external auditory canal. Dr. Sutphen stated at this time the patient was slightly tender

in front of the left ear. He was of the opinion that the patient was developing a furuncle of external canal. The patient's hearing at this time was very good; there was no dizziness, no headache, no tenderness or redness over the old scar.

On the afternoon of Oct. 21, the patient felt well but in the afternoon traveled by automobile and was exposed to cold and dampness. During the night the left ear began to discharge. Oct. 22 consulted Dr. C. H. Schlichter and myself. Temperature 101°. Patient did not appear sick; no tenderness over the mastoid. On going home patient felt chilly and after reaching home had a slight chill. Was seen by Dr. Sutphen that night (October 25, 1917). Rales found over the base of the right lung. Patient complained of a slight cough and said his head hurt when he coughed. Temperature 102° F. Patient appeared slightly drowsy, although no importance was attached to this symptom at that time.

The following morning (Oct. 23, 1917) the patient was markedly drowsy; hearing was good; temperature 104° F. Dr. Thatcher was called and double pneumonia dianosed, the cerebral symptoms being ascribed to the pneumonia.

Patient had a fairly good day. That night had rythmic movements of his right hand. At 5 o'clock a. m. Wednesday, Oct. 24, suddenly had a convulsion, which was followed by others of greater severity as the day went on.

When seen by me Oct. 24 he was in deep coma; flaccidity of right arm and leg; no Babinski on the right; rigidity of the neck; convergent strabismus of the right eye; left optic disc was red and the veins full; pupils small; right fundus not seen on account of small pupil and poor illumination; pulse ranged from 98 to 130; temperature between 102 and 104° F.; ear did not show much discharge, and there was no tenderness or redness over the site of the old operation. A lumbar puncture showed flocculent spinal fluid, not under great tension, the examination of which showed pneumococci in large numbers; 2,000 cells to each field. Blood count 29,000 white cells. Death in deep coma on the following morning.

Comment: There is little doubt in my opinion that the terminal pneumococcic infection was a recrudescence from the vaccine treatment and exposure of the original infection, although I am willing to admit this must be more or less speculative. Still, while in the service, I witnessed so many cases of recrudescence of latent infection from vaccine treatment that to my mind the evidence warrants the placing of of the above case in this group.

II—(c) *Local Exudative Type*: Of course it is possible to have a localized exudation into the sub-arachnoid spaces, but this generally follows by direct extension from an adjacent focus of suppuration; as an intra-piarachnoid abscess located around the internal auditory meatus from involvement of the labyrinth, or over the tegmen, by direct extension from the middle ear. An intra-piarachnoid abscess, however, is generally the result of the protective meningitis becoming infected from the adjacent focus of infection and is but very rarely the result of *localization of a general meningeal infection.*

III. *Protective Meningitis*—pure and simple—pathologically characterized by exudation in the sub-arachnoid spaces, which exudate, however, is *sterile.* In this type the clinical cerebral manifestations are the result of increased production of cerebro-spinal fluid and of blocking the pathways of the cerebro-spinal fluid sys-

tem, in the effort of the protective meningeal mechanism to prevent the infection of the sub-arachnoid space from a neighboring bacterial or toxic irritant.

The protective type may be (a), local—(a very frequent occurrence)—or (b), general.

The clinical symptoms of a simple protective meningitis—a "sympathetic meningitis"—may be of the severest kind. toxic and compressive, with high temperature, delirium, coma, or convulsions, and aphasia. Lumbar puncture in the general type reveals a cloudy sterile fluid, a very high cell count, an increase in the globulin content, the Fehling reducing agent continuing present however; while in the local type the fluid—though increased in amount—may be clear and without a high cell count.

If the exciting cause of the protective meningitis is an adjacent focus of infection in the ear or nose, its removal by operation before the actual invasion of the sub-arachnoid spaces by bacteria has occurred will immediately be followed by a disappearance of all the symptoms; especially, if associated with an evacuation of the contiguous, localized mass of cerebro-spinal fluid; but if the localized collection of cerebro-spinal fluid becomes infected—from a direct extension of the suppurative process—an intra-piarachnoid abscess follows.

In an examination of all the cases of suppurative meningitis, I am impressed with the number of cases so classified which in reality are but localized piarachnoid collections of pus (from labyrinthitis, necrosis of dura, or brain abscess) with a complicating general protective meningitis, as shown by the abscence of micro-organisms in the cerebro-spinal fluid obtained by lumbar puncture. Of course, with the evacuation of the localized irritant, combined with frequent lumbar punctures, the fluid rapidly clears.

The majority, if not all of the cases of Kading (cases 1 and 2), Emerson, Witzel, Schlesinger, Lucae, Kummell (1905), Jansen and Day (case 2), although recorded as recoveries from suppurative meningitis, probably belong to this group.[27 34]

Case IV. Case of Generalized Protective Meningitis, Originating from Labyrinthine Suppuration, with Symptoms of General Suppurative Meningitis—High Temperature, Headache, Stiff Neck, Kernig, Cloudy Spinal Fluid, Increased Globulin, but Without Micro-Organisms, the Sugar Content Remaining Present—Absolute Disappearance of all Symptoms on Discharge of Small Squestra of Cochlea.

J. G.; Male; Adult: Seen with Dr. S. H. Baldwin, November 24.

History: Had had discharging right ear for three months. For last three weeks had pain in right ear with severe pain in head, in right temporal region, and dizziness. The night before, slight facial paralysis.

Examination: Right drum membrane destroyed; granulations and pus in external canal. No hearing in right ear.

Rotation to Right: Horizontal nystagmus to left of 9 sec. No dizziness or nausea. Wide deviation of right to right (10 inches). Slight deviation of left to right (3 inches).

Rotation to Left: Horizontal nystagmus to right of 5 seconds. No dizziness or nausea. No deviation of either hand.

Cold Caloric in Left: Produced no nystagmus—if any it was horizontal to left; no pointing deviation of either hand; no dizziness or nausea. Throwing Head Back: Produced a violent horizontal nystagmus to right; quick movement and large amplitude; wide pointing deviation of both hands to left; dizziness.

Head Forward, Rotating to Right: On straightening up, fell to right; dizziness and nausea.

Head Forward—Rotating to Left: Did not fall; very slight dizziness. Temperature, 99°; Pulse 76.

November 27: Lumbar puncture performed; fluid clear, under pressure. Globulin increased. Fehling's slightly reduced. Cell count 100 cm. No organisms.

November 28: Lumbar puncture; fluid clear, under pressure; globulin increased; sugar reduction normal; no organisms.

November 29: *Operation*: Labyrinth opened; fistula found; granulations. Posterior fossa opened behind posterior pyramid; quantity of cloudy cerebro-spinal evacuated. Following operation patient had severe pain over left eye; numbness of fingers of left hand; was restless at night; temperature 101-103°; Leucocytes count 12,800; Polynuclears 73%.

December 3: Stiff neck; severe pain in head; Leucocytes count 16,400, Polynuclears 85%. Smear from wound: Staphylococcus, streptococcus mucosus capsulation Gram + bacilli. Temperature 102-104°. Pulse, *slow*, 82-90.

From December 3 to December 28 patient continued with restlessness at night, slightly delirious with alternating attacks of stupor from which he could easily be aroused; severe pain in the head; stiffness of neck; Kernig; pain behind left eye; photophobia, with a continuously high temperature—101-105°—and a proportionally slow pulse. Several blood cultures were negative. Leucocytosis.

December 26: Lumbar puncture; no pressure; color *pale yellow;* globulin greatly increased; sugar reduction about one-half normal; cell count 633 cm.

December 31: Patient seemed better in every way. Temperature 99-101°.

January 7: Patient looked very sick; pulse very fast and of poor quality, but he rested better at night. Still complained of stiffness in back and legs, but head felt better.

January 18: Patient much better in every way; temperature normal.

February 24: Patient discharged from hospital feeling fairly well, but with a discharging ear. A few days later discharged a sequestra of cochlea, after which the ear promptly healed and all symptoms disappeared.

A similar case, with dead labyrinth, high temperature, delirium, almost maniacal headache, double papilloedema, cloudy spinal fluid, with cell count of over 3000 but without micro-organisms, an exenteration of the labyrinth, with incision of the dura, was promptly followed by recovery.

Case V. Case of Localized Protective Meningitis, Result of Adjacent, Quiescent Abscess in Petrous Pyramid—With Healed Mastoid Wound and Middle Ear.

Boy; referred by Dr. A. G. Howe, Brooklyn, February 4th.

History: His tonsils were removed about five years ago. He had always complained of aching or weakness in his legs. For past few years had earache in winter; the mother is not sure which ear was affected, but thinks it was the same ear which is now troubling him

—the right ear. There was no discharge in the beginning; the ear apparently being dry. Last winter a physician punctured the ear—no discharge until a similar puncture this year by Dr. Howe; the ear then discharged, and, as the pain did not cease, Dr. Howe performed a mastoid operation.

Boy was very restless at night before his operation. Following the operation he was very much better for several days. Then he had two chills, not very marked, however. Since then he did not seem well; constant headache and a marked tendency to sleep. The mastoid wound was entirely healed.

February 4: Boy complained of pain over and through the right eye; felt very weak and drowsy; did not want to eat because he had "a full feeling in his stomach"; says his legs or knees hurt and felt weak if he tried to walk. If he looked at anything his eyes hurt, but he thought he saw well. His temperature was not above 100° since mastoid operation.

Outstanding Symptoms: Severe headache—right frontal—very severe at night following mastoid operation three weeks ago. Drowsiness, weakness, aching in legs, aching in right eye, especially when fixed.

Examination: The boy looked sick and had a tendency to hold his head forward. The left pupil slightly larger than the right, but this might have been because drops were put in while he was in the hospital. Ophthalmoscopic examination showed veins on left eye slightly larger than in right, and both slightly congested. Both vision—20/20.

Hearing:— Right Ear: Watch—one foot; Whisper—across room; Left Ear: Watch—five feet; Whisper—across room;

Vestibular Reactions:— No spontaneous past-pointing; No spontaneous nystagmus.

Rotation to right:—Horizontal nystagmus to left of 24 sec.; Dizziness; no nausea; Wide deviation of both to right.

Rotation to Left:—Horizontal nystagmus to right of 19 sec.; Dizziness; no nausea; *No deviation of either hand.*

Cold Caloric; Right:—(Affected side)

Right Ear

Head erect; Nystagmus, rotary, to left—good amplitude. Deviation of both to right; Dizziness; no nausea.

Head back; Nystagmus, rotary, to left; Wide deviation of both to right; No increase of dizziness; no nausea.

Left Ear (Good ear)

Head erect; Slight horizontal nystagmus with rotary tendency to right; No deviation of either; Dizziness; nausea.

Urine, 1032; no sugar; no albumen.

Lumbar puncture, February 5: Spinal fluid, amount 3 cc.; fluid bloody; globulin slightly increased; sugar normal; no oragnisms in smear; culture negative after 48 hours; no cell count, owing to presence of blood.

February 5th to 10th: Temperature ranged from normal to 102.2.

Impression: "Patient must have either an extra-dural abscess, an intra-dural abscess, or serous meningitis. I do not think it can be sinus thrombosis. It looks like an intra-dural abscess; but not in the cerebellum. It must be in the temporsphenoidal lobe."

***Operation*: February 10: Incision directly above the ear, through the temporal muscle and fascia; rongeured away the bone; saw the posterior branch of the middle meningeal. Just above this made a nick in the dura; a large quantity of cerebro-spinal fluid evacuated, which apparently was clear and under tension. Exploration within the substance of the brain failed to reveal pus. The mastoid wound, which had been closed, was now re-opened, and found to contain, deep in, under the antrum, a pocket of pus. Posterior fossa exposed; middle fossa exposed.**

***Comments on Case*:**

There can be no doubt that this boy had a serous meningitis, the result of extra-dural abscess. This would account for the pain behind his eyes; the disproportion of the symptoms since operated on in Brooklyn; and also, for his not having cerebro-spinal fluid under pressure at lumbar puncture, the serous meningitis being local in character.

VALUE OF AN EXISTENT PROTECTIVE MENINGITIS AT TIME OF BACTERIAL INVASION OF CEREBRO-SPINAL FLUID SYSTEM.

Even when the general sub-arachnoid space has actually become involved in the bacterial invasion, as in meningitis following the evacuation of a brain abscess—the infection of the general sub-arachnoid space being shown by the presence of bacteria free within the cerebro-spinal fluid—*if the protective forces of the meninges have been previously fully marshalled*, the infection may still be limited providing drainage of the infected area is established. (See Case IX, at the end of this article.)

Of prime importance in the treatment of meningitis is the realization that all four types pass freely from one to another. The hope of attacking the problem scientifically lies in an attempt at a correct differentation, and in the adoption of measures to combat the pathological condition present at the moment.

It is a clinical fact of consequence, and one to which attention previously has not been directed, that the presence of a protective meningitis induced by a suppurative process within the cerebral substance itself (such as a brain abscess; or suppuration within an adjacent structure in communication with the sub-arachnoid space, a suppurative labyrinthitis, gangrene of the dura or an intra-pirachnoid abscess), is the chief factor in the cure of a generalized suppurative meningitis. An examination of the recorded recoveries from a general suppurative meningitis—exclusive of those of meningococci origin—demonstrates that in fully two-thirds of the cases which have recovered there was present a protective localized meningitis already in action *at the time of the general invasion* of the sub-arachnoid spaces by the infecting micro-organisms; the pathological processes found at operation being such as necessarily are associated with a previous protective meningeal inflammation.

INDICATIONS FOR THE SELECTION OF INDIVIDUAL METHODS OF TREATMENT.

What then, we must ask, are the indications for treatment, and what the therapeutic means at our disposal? First it must be recognized that no *one* therapeutic agent or operation will solve the problem of meningitis. Hope lies in the application of the proper therapeutic agent to the *individual case* at the *specific moment.* For a measure such as irrigation of the sub-arachnoid space which at one moment may be of avail by removing the cause of infection, subsequently may become impossible because of the filling of the spaces, or valueless because of the extension of the infection into the ventricles.

Pathological Factors to Be Considered: Four factors enter into the consideration of treatment:

(1)—The virulence of the micro-organism when implanted in the sub-arachnoid spaces.

(2)—The protective reaction of the tissues of the sub-arachnoid spaces to the invading irritant.

(3)—The effect upon the brain of closure of the cerebro-spinal fluid circulatory system—(a) by interfering with its proper nutrition, and (b) by causing compression.

(4)—The effect of the infection on the general vascular circulation by the emptying of its bacteria and toxines into it.

If the virulence of the infecting organism in the tissues of the sub-arachnoid space is low, and the protective tissue reaction prompt, (as is the case in epidemic cerebro-spinal meningitis) recovery may follow, either spontaneously, or after the injection of a specific serum (meningococcic)—which doubtless assists, not only by killing the micro- organism, but also by stimulating the protective reactive mechanism.

With a high degree of virulence and a poor protective tissue reaction the prognosis must be unfavorable. Sub-arachnoid irrigation, however, would logically appear to hold out some hope for recovery, by removing the bacteria and exudate and stimulating the protective reactive forces.[35]

If, however, the protective reactions of the meninges have already been well established at the time of the infection—as in meningitis following brain abscess, necrosis of the dura, labyrinthitis, and so on—the prognosis is far from hopeless; especially if the suregon drains the original source of infection. In addition to this, I believe the pathological condition within the cerebro-spinal circulatory system

calls for sub-arachnoid irrigation to remove the infecting micro-organisms, to keep open the cerebro-spinal fluid pathways, and further to stimulate the protective meningeal reactions.

Treatment:

(1)—Eradicate or drain known foci of infection, such as mastoid, nasal accessory sinus, or bony cranial involvement, extra-dural abscess, sinus thrombosis or labyrinthitis, intra-cerebral abscess, intra-piarachnoid abscess, or localized piarachnoid collection of cerebro-spinal fluid;

(2)—Free the pathways of the cerebro-spinal fluid circulatory system by sub-arachnoid drainage, thus partly at least, removing the micro-organisms and exudates, and attempting to prevent occulusion of the foramina of Luschka and Magendie, and extension into the ventricles, with associated cerebral compression;

(3)—Encourage the development of the protective reactions of the meningeal mechanism by the injection of non-toxic serums into the sub-arachnoid spaces;

(4)—Combat general blood-stream sepsis by Murphy drip, diuresis, diet, et cetera;

(5)—Finally, control cerebral compression when present.

RELATIVE IMPORTANCE OF THERAPEUTIC MEASURES.

Logically and clinically, (from an examination of the recorded cases of recovery from suppurative meningitis previously mentioned), the most important indication is the evacuation of any causative localized intra-dural focus of suppuration—an intra-cerebral or intra-piarachnoid abscess labyrinthitis, or infection from a bony trauma. (See Case VIII, end of this article).

In the presence of such a lesion the number of recorded recoveries is so few—only twenty-two in all literature in spite of the large number of operations performed—that I am of the opinion that sub-arachnoid irrigation should immediately be instituted. In the absence of a localized focus of infection, either within or connected with the dura, the clinical evidence of the few recorded cases of recovery in this type of the disease— only eight in the literature in spite of the immense number of operations—would compel us, I think, to regard the usual operative procedure for drainage as not warranted; especially as, in addition to the clinical evidence, the post-mortems of Day[36] have demonstrated that drainage operations are pathologically inadequate to control the disease.

In those cases, then, in which there is no demonstrable diagnostic or operable lesion within the brain or directly involving the sub-

arachnoid spaces, sub-arachnoid irrigation should be instituted at the earliest possible moment, since up to the present, this holds the only logical hope of recovery.

An understanding of the subject will be assisted by a study of the relative frequency of the paths of infection in otitic meningitis as found "either on clinical or on operative examination, or on the basis of autopsy findings," as reported by Breggren[37] (90 cases) and Mygind (35 cases).

	Berggren	Mygind	Total	
Labyrinth	27	11	38	30%
Sinus Phlebitis	28	6	34	27.2%
Pachy-meningitis (with or without brain abscess)	15	2	17	13.5%
Through Osteitic Changes (Vault)	4	7	11	8.8%
Untraceable	16	9	25	20%
	90	35	125	

If we combine the statistics of Berggren and Mygind, we have 125 cases, in which, in 100—or in 80%—a path of infection was traceable and in 25—or 20%—the path of infection was untraceable. Judging from this, in otitic suppurative meningitis, an operation which attacks the original source of infection is indicated in about 80% of the cases.

Types of Meningitis Calling for Sub-Arachnoid Irrigation;

It is to be remembered that many cases of pneumococcus meningitis of aural origin clinically present but few symptoms for a considerable period of time after the appearance of micro-organisms in the cerebro-spinal fluid—a feeling of "not well," with more or less headache, being the only complaint[38] (Berens, Day, Kopetsky).

As all of these cases, up to the present, have ended fatally, it would seem to me that irrigation of the sub-arachnoid spaces is especially indicated. This method logically holds out a possibility of cure by eliminating a part of the infection, keeping open the cerebro-spinal fluid pathways by stimulating the meningeal protective mechanism —which, for some unaccountable reason, appears more or less quiescent—the more so as the end frequently comes suddenly, life ceasing because its "governing, controlling, and correlating centers are entirely prevented from continuing their functional activity,"[39] possibly from a blocking of the cerebro-spinal fluid circulatory system.

Again, as previously described, the pathological picture of human fulminating pneumococcus meningitis, type III, is so similar to that seen in experimentally induced fulminating meningitis from blood-stream infection—punctate hemorrhages in the piarachnoid, followed by an exudation at first confined to the spaces over the cortex—that one is forced to regard it as identical; *excepting that in man the aural or nasal suppuration precedes the meningeal involvement.*

If the contention made in this paper be true: that the human type of fulminating meningitis is at first strictly a local process (the sudden meningeal involvement being possibly a combination of local blood-stream infection, plus a disturbance of the meningeal protective mechanism, and *later* a general blood-stream infection) then this type offers a possibility of cure by early lavage of the sub-arachnoid spaces. Especially as clinical experience has demonstrated that in the early stages few bacteria are obtainable from the cerebro-spinal fluid, and experimentally at this time the infection is limited to the sub-arachnoid spaces of the cortex.

The inferred therapeutic indications, then, in the fulminating type of suppurative meningitis—pneumococcus meningitis—would be the early removal of the bacteria and the stimulation of the protective meningeal mechanism *before the invasion of the ventricles.* In the acute, sub-acute and chronic exudative types—streptococcic and meningococcic—the same course should be pursued to prevent the exudate from filling the sub-arachnoid spaces, causing obliteration of the peri-vascular spaces and extension into the ventricles.

Cerebral and Cerebro-Spinal Sub-Arachnoid Irrigation:

Historical and Clinical: Roth, in 1893, suggested irrigation of the sub-arachnoid spaces, from the Sylvian region to the occipital regions of the base with boric acid solution.[40] Barr experimentally demonstrated that fluid irrigation into the lateral ventricles would be recovered from the lumbar region, and in its course permeate the subarachnoid spaces of the cortex and the basal cisterna. He also practiced irrigation of salt solution on a patient in extremis, the patient living fourteen hours[41].

Following the suggestion of Barr, Day unsuccessfully attempted to irrigate through the ventricle into the lumbar region. He also washed out the spinal canal with normal salt solution passed from a canula into the lumbar region through an operative opening in the cisterna magnum.[42]

Cushing demonstrated that normal salt solution without a calcium content had a toxic influence on the cerebral tissues.[43]

Wegeforth and Essick, and Weed and Wegeforth first experimentally demonstrated that lethal action of all antiseptics. When *irrigated through* the sub-arachnoid space even sterile, distilled water is highly toxic, while a saline solution without a proper proportion of calcium frequently was followed by a fatal respiratory or circulatory embarrassment. Later they demonstrated that a properly balanced, modified Ringer's solution may be passed through the sub-arachnoid space with safety.[44]

It is to be understood that *irrigation of* and *injection into* the sub-arachnoid spaces are fundamentally different. Injections limited in extent may not be lethal, because of the limited degree of toxemia which they may cause. Irrigation fluids to be safe must be balanced to a nicety as to composition, and approximately of the body temperature.

It would seem that the early experiments of Falkenhein and Nauryn[45] and L. Hill[46] did not have a lethal effect because they simply injected

sodium chloride solution and did not irrigate it through the spaces. Franca[47] and Wolff[48] both partially irrigated the *spinal canal* with lysol and proto-argol in meningococcus meningitis, the latter reporting five cases of recovery, after failure with serum alone. The immunity of the Franca and Wolff cases from lethal toxic effects was because both of these observers did not, in the true sense, irrigate the sub-arachnoid space of the spinal cord—they simply injected small quantities of fluid after allowing the flow of cerebro-spinal fluid. Franca injected lysol into the spinal canal, one needle being in the twelfth dorsal space, the other in the lumbar secral space, through these he passed the solution of lysol, using in adults from 12-20 cc., and in children, 3-9 cc., while Wolff injected 10 cc. of saline solution into the lumbar region. The method of Franca and Wolff is spinal *injection*. Cerebral or cerebro-spinal *irrigation* of such toxic substances would of necessity be rapidly fatal.

A careful review of the recent literature, while disclosing many cases of so-called lavage in epidemic cerebro-spinal meningitis, fails to disclose any of true sub-arachnoid irrigation.

Thus Farmachides[49] simply injected salt solution into the spinal canal after a lumbar puncture, and repeated the process; and Kading[50] also injected the lumbar sac with fluid, although he called it "lavage."

Effect of Cerebro-Spinal Sub-Arachnoid Irrigation of a "Properly Balanced Physiological Solution" on the Chemical Composition of Cerebro-Spinal Fluid.

Weed and Wegeforth[51] experimentally demonstrated that the "sodium chloride and the globulin content and gold reaction were unaffected after irrigation with a properly balanced physiological solution thus showing absence of irritation. However, irrigation with any antiseptic solution, even in great dilution, was followed by enormous increase in the cell count. This persisted from five to six days, according to Wegeforth and Essick,[52] at the end of which time, although the cerebro-spinal fluid showed a return to normalcy, the animal became worse.

Extent of Area Involved in Irrigation:

Barr,[53] Weed and Wegeforth[54] found experimentally that a cerebral sub-arachnoid irrigation of a pigment was followed by the uniform distribution of the pigment over the major part of the homolateral hemisphere, the contra-lateral hemisphere being covered about two-thirds, and *the basal cisterna filled;* also, they found "that spinal irrigation resulted in the whole spinal arachnoid space being pigmented, and this extended into the basal cisterna." This experimental evidence coincides with my own clinical experience—that the removal of the waste material lessens the toxemia, although it may not completely control the infection.

In the Weed and Wegeforth[55] experiments in cerebral and spinal irrigation two of the animals with experimentally induced meningitis recovered, and the prolongation of life in all so treated was apparent. In those that did not recover the "washing away of some of the toxic products of bacterial growth seemed also of service for the irriagted animal was usually more nearly normal for the next six hours than the controls." In these experiments multiple irrigation was not attempted, which my experience with Case VI would make me think absolutely essential.

Operative Technic:

Incision of the dura may simply open the sub-dural space; but the in-flow canula must be introduced *into the sub-arachnoid spaces,* as otherwise the irrigation may distend the sub-dural space and have no effect—a condition which probably occurred in one of

my irrigations. Another graduated needle of rather large bore is introduced into the cerebello-medullary cisterna—following the occipito-atlantal puncture technique of Ayer—and recovers the outflow.

Amount of Fluid: The amount of fluid possible to pass through the cerebral and spinal arachnoid spaces varies. Weed and Wegeforth[56] state, "In *spinal* irrigation in normal animals the flow was always slow, and a large amount of fluid was not obtainable at the needle of exit," . . . . "only 106 cc. after one and one-half hours." "In normal animals the time of *cerebral* irrigation was one hour, and the amount of fluid recovered varied from a few hundred to over one thousand cc." In the Weed and Wegeforth experiments in infected meninges, cerebral irrigation was undertaken in two or four hours after infection, and from fifty to five hundred cc. irrigated in thirty minutes—that is, about one and three-fourths to seventeen cc. per minute. This experimentally demonstrated how the presence of the pathological exudate in the sub-arachnoid space interferes with the free lavage possible in normal meninges. All of my own cases demonstrate that if this method is to be fully successful it must be undertaken early in the disease.

Speed of Flow: In all of my three cases of cerebral irrigation the flow was somewhat slow, and in the one, with a great deal of exudate, the exit needle constantly was becoming clogged with shreds, necessitating its frequent cleansing with a stylette. Also, at the point of entrance, distension of the sub-arachnoid space frequently occured. Consequently it is suggested that several in-flow openings be made and that the irrigation, when not free through one, be stopped and another opening be used, as in Case VIII, reported at the end of this paper.

It is evident that the free, continuous stream which can be obtained experimentally in a normal animal[57] is not possible after the sub-arachnoid spaces have become the seat of an exudate. It is advisable in otitic cases to introduce an exit canula into the lateral cisterna as well as into the occipito-atlantal and lumbar regions, and not, as in my first case, to attempt to irrigate from the lateral cisterna through to the lumbar region. I am persuaded that the larger the number of openings made both for entrance and exit the better the prospect of accomplishing the object.[58]

Maintenance of Even Temperature: An even temperature of ninety-nine is maintained throughout the operation by an outer water-bath for both container and tubing. In both water-baths a thermometer is kept constantly; to regulate this the constant attention of an assistant will be required who has had some experience in a chemical laboratory. His sole duty should be (a), to regulate the temperature of the in-going fluid and (b), to note and record the rate and amount of the in-flow. Another assistant notes and records the rate and amount of the recovered fluid, and at the same

time holds the canula in the occipito-atlantal and lumbar regions. The operator himself should place and hold steady the in-take canula within the sub-arachnoid space.

CONCLUSIONS.

Irrigation of the sub-arachnoid spaces keeps open the tracts for the normal circulation of the cerebro-spinal fluid, essential to the removal of waste products from the cerebral tissue substance, if not for its actual nutrition. It also, partially, at least, removes the bacteria and exudates produced by the bacterial and toxic reactions of the tissues themselves.

My experience with the three cases reported, leads me to assert that cerebral irrigation of the sub-arachnoid space is a painless proceeding, without great danger if conducted with scrupulous care as to technic; and as an examination of all the recorded cases has demonstrated that suppurative meningitis is almost invariably fatal, I am of the opinion that immediately upon the demonstration by lumbar puncture of micro-organisms—exclusive of the meningococcus group—in the cerebro-spinal fluid, a cerebral and cerebro-spinal sub-arachnoid irrigation should be instituted.

ANALYSIS OF THIRTY-ONE COLLECTED CASES OF RECOVERY FROM GENERAL SUPPURATIVE MENINGITIS:

In an examination of the literature I can find only thirty cases of recovery from general suppurative meningitis in which the diagnosis was substantiated by the finding of micro-organisms in the cerebro-spinal fluid; these with the one herein recorded case, make a total of thirty-one recoveries from a general suppurative meningitis.

Of the collected cases two are simply referred to by Du Bois and Neal—"Summary of Seven Years of Clinical and Laboratory Epperience with Meningitis in New York City," *Department of Health Reprint Series*, No. 62, September, 1917—"No patient with pneumococcus meningitis directly under our supervision has recovered, but we know of two patients that did." These two cases consequently can be utilized only in a consideration of the kind of infecting micro-organism from which recovery is possible.

Of the remaining twenty-nine, two—Gobell's and Kummell's—are very superficially recorded, having been orally reported at a meeting of the Society of North West German Surgeons, and published only in abstract in the *Centralblatt fur Chirugie*, 1909, Vol. XXXVI, No. 34, p. 1179.

Of the remaining twenty-seven, two—Tedesco's and Netter's—are lacking in the details essential to a proper analysis. Tedesco—"Streptokken Meningitis durch Lumbalpunktion geheilt;" *International Centralblatt fur Ohrenheilkunde,* 1910-'11, Vol. IX, p. 365—makes no mention of the cause of the meningitis, and Netter—"Meningitis cerebro-spinales suppurees a streptocoques consecutives a la scarlatine;" *Bull. et Mem., Soc. Med. des Hop. de Paris,* 1909, Vol. XXVII, p. 11—presents his case primarily to demonstrate the value of lumbar puncture, but unfortunately fails to enumerate many details necessary to complete analysis.

Of the twenty-four cases still to be considered, one—Gradenigo: "Uber die Diagnose und Heilbarkeit der otologischen Leptomeningites;" *Archiv. fur Ohrenheilkunde,* 1899, Vol. 47, p. 158—in its history, course and outcome warrants the suspicion that it primarily was a protective meningitis, the micro-organisms found in the cerebro-spinal fluid by puncture being possibly a contamination from the skin. In one—Held, R. J. and Kopetsky, Samuel J.: "Report of a Case of Purulent Meningitis Following Radical Mastoid Operation; Recovery after Operative Interference;" *Archives of Otology,* 1906, Vol. XXXV, p. 531—although fully reported and discussed is not above the suspicion of being a meningococcic infection. This is doubly true of my own case—Case VII—my own being an intracellular diplococcus complicating a fracture of the skull, and Held and Kopetsky's a diplococcic meningococcic infection engrafted upon an otitis media with an extra-dural abscess followed later by a mixed infection possibly from contamination during the course of the disease. This suspicion is further justified, in Held and Kopetsky's case, by the very high cell count—310,000—in the cerebro-spinal fluid; a condition which, although frequent in meningococcus meningitis, is most unusual in the cases of recovery from other types of micro-organism.

Utilizing all the data, the analysis must vary between thirty-one and twenty-five cases of general suppurative meningitis which have been cured.

TOTAL NUMBER OF COLLECTED CASES AVAILABLE FOR ANALYSIS—[59]

KAUSCH: Die Behandlung des Hydrocephalus mit Konsequentis. Punction; *Mittheil. aus den Grenzgeb, des Medizin and Chirurgie,* 1910, Vol. 21, p. 329, Case III.

HELD and KOPETSKY: Loc. cit.

GROSSMAN, F.: Kasuistisches zur Lumbalpunktion und Circumscripten Meningitis; *Archiv fur Ohrenheilkunde,* 1905, Vol. LXIV, p. 24.

BECK, J. C.: Failures and Successes in Diagnosis and Surgical Intervention of Some Intracranial Diseases, Especially from the Stand-

point of an oto-laryngologist, with Report of Cases; *Illinois Medical Journal*, 1913, Vol. XXIV, p. 265.

BONDY—three cases: Zur Frage der Heilbarkeit der otogenen Streptococcen Meningitis; *Wiener Medizinger Wochenschrift*, 1917, Vol. LXVII, p. 1600.

DUBOIS PHEBE L. and NEAL, JOSEPHINE B.: Loc. cit.

WATSON WILLIAMS, P.: Case of Labyrinthitis, Diffuse Purulent Meningitis; Labyrinthotomy; Recovery with Intra-venous and Intra-thecal Injections of Colloidal Silver; *Journal Larynology, Rhinology and Otology*, 1920, Vol. 35, p. 197. Also *Trans. Royal Soc. of Medicine; Section of Otology*, 1920, p. 73.

POIRIER: Fracture de l'etage anterior du crane, meningite consecutive, trepanation double, guerison; *Bulletins et Memories de la Societie de Chirurgie*, 1901, Vol. XXVII, p. 17.

ALEXANDER, G.: Klinische Studien zur Chirurgie der otogenen Meningitis; *Archiv fur Ohrenheilkunde*, 1908, Vol. LXXVI, p. 1

NETTER, A.: Loc. cit.

KOSTLIVY, S.: Die Operation der eiterigen Meningitis; *Archiv fur Klinischer Chirurgie*, 1912, Vol. 97, p. 627.

GOBELL: Loc. cit.

TEDSCO: Loc. cit.

DAY, EWING W.—three cases: Indications for and Results of Operative treatment of Otitic Meningitis; *Surgery, Gynecology and Obstetrics*, April, 1913, pp. 369-376.

KUMMELL: Loc. cit.

LITCHFIELD—five cases: Pneumococcus Meningitis: Treatment by a Specific Antipneumococcus serum; *Journal American Medical Association*, May 10, 1919, p. 1345.

HELD, R. J., and KOPETSKY, SAMUEL J.: Loc. cit.

GRADENIGO: Loc cit.

LEWKOWICZ, K.: Guerison per un vaccin specifique d'un absces cerebral et d'une meningite generale a staphylocoques, consecutifs a un coup de feu crane; *Archiv de Med. des Enfants*, 1920, Vol. XXIII, p. 540.

O'MALLEY, J. F.: Case of Purulent Otitic Meningitis; Operation; Recovery; *Transactions Royal Society of Medicine*, February 20, 1920, p. 72.

SCOTT, SYDNEY: Streptococcal Leptomeningitis in a child due to Chronic Suppurative Otitis Media; Rapid Development of Coma; Radical Mastoid Operation with Translabyrinthine and Lumbar Thecal Drainage with Complete Recovery; *Journal of Laryngology and Otology*, July, 1921, Vol. XXXVI, No. 7, p. 356.

EAGLETON, WELLS P.: See Case VI, at end of paper.

PRIMARY FOCUS OF INFECTION.

Of the thirty-one collected cases of recovery the primary focus of infection was:

(a)—From an otitis media, twelve cases—Beck, Bondy (three cases), Netter, Du Bois and Neal, Held and Kopetsky, Day (three cases), Gradenigo, and O'Malley.

(b)—From a labyrinthitis following an otitis, four cases—Watson—Williams, Alexander, Grossman, and Scott.

(c)—From a labyrinthitis precipitated by a trauma, one case—Bondy.

(d)—From an otitis precipitated by a trauma, one case—Kostlivy.

(e)—From a perforating trauma of the dura, three cases—Kausch, Poirier and Lewkowicz.

(f)—From a fracture of the skull, with hemorrhage from the ear and nose, (and consequently may be regarded as conpound although the hearing was not destroyed), one case—Eagleton.

(g)—From, or concurrent with pneumonia, (and consequently may be regarded as of blood-stream origin), five cases—Litchfield.

(h) Primary focus not stated, four cases—Kummell, staphylococcus albus; Gobell, staphylococcus aureus; two cases referred to by Du Bois and Neal, pneumococcus.

Protective inflammation Present Prior to Development of General Meningitis and acting as chief curative agent.

Twenty-six cases are recorded with sufficient detail to warrant an attempt to analyse the pathological lesion present at the time of the invasion of the general cerebro-spinal fluid system. Of these, five—Litchfield's—or—19 %, were undoubtedly of blood-stream origin, the pneumococcus meningitis originating from a pulmonary infection, although frequently the meningeal symptoms at first overshadowed the lung manifestations. The remaining 21 cases —or 81%—include all that were *preceded by* a local infection. Of these twenty-one, the injury or an operation disclosed a *local pathological* process necessarily *associated with a previous protective* meningeal reaction in fourteen, and probably in sixteen cases—or 70% and 80% of all cases of local origin.

Mygind's[60] well known report of cure in six cases unfortunately cannot be included on account of incompleteness of details, especially as to the presence of micro-organisms in the lumbar puncture fluid; he evidently makes no distinction between general suppurative meningitis and a protective meningitis. He states, "Bacteria are generally present; when they are absent the prognosis is favorable." The one case fully reported undoubtedly was entirely protective as "the lumbar puncture yielded a very turbid fluid which contained numerous polynuclear cells, but no bacteria."

From the wording of the report all the other five cases presumably were preceded by a protective meningitis, as they all apparently followed brain abscess, sub-dural abscess, labyrinthitis, necrosis of the dura, or sinus thrombosis. These five cases, if included in the analysis, would, of course, considerably increase the proportion of cures of meningitis of local origin in which a protective meningitis reacton was present prior to the invasion by micro-organisms.

DETAILS OF LESION PRESENT IN SIXTEEN COLLECTED CASES OF RECOVERY FROM GENERAL SUPPURATIVE MENINGITIS OF LOCAL ORIGIN THAT NECESSARILY WERE PROCEDED BY A PROTECTIVE MENINGITIS EITHER LOCAL OR GENERAL.

(a)—Preceded by a localized, intra-dura suppuration; and consequently associated with a more or less general protective menin-

geal reaction—six cases; of these, two were preceded by a brain abscess, (Day), treated by Haines' operation; one was associated with or preceded by an intra-piarachnoid abscess—otitic—(Held and Kopetsky), treated by drainage; and three were associated with or preceded by a sub-dural, traumatic abscess, (Kostlivy, Poirier and Lewowicz), treated by drainage.

(b)—Preceded by a local protective meningitis of local extra-dural origin—eight cases; of these, five were preceded by a labyrinthitis—the Bondy and Alexander case, treated by a labyrinth operation; the Watson-Williams case, also treated by a labyrinth operation and intra-thecal and inter-venous serum; the Grossman case, treated by a radical operation; the Scott case, treated by trans-labyrinthine drainage.

One case associated with a perforating, infective traumatism, (Kausch), treated by drainage; one associated with necrosis of the dura, (Bondy, Case II), treated by dural incision; and one, following a compound fracture of the skull, (Eagleton), treated by sub-arachnoid irrigation and intra-spinous injection of meningococcic serum.

(c)—Probably preceded by a protective meningeal reaction—pachymeningitis interna and local piarachnoid exudate—two cases; one following a sinus thrombosis, (Beck), treated by Haines' operation; the other, showing Gradenigo's syndrome, in which probably there was a local pachymeningitis or a local protective piarachnoid collection of fluid over an infected area of bony caries deep in the petrous pyramid, (Bondy Case III), treated by a mastoid operation.

In fifteen of these sixteen cases an operation by eradicating or draining the causative, intra-dural irritant may be regarded as the chief, but not sole factor in the recovery. The operation simply broke the vicious circle and eliminated the causative factor, thus allowing the already active protective meningeal mechanism to combat the micro-organisms which had gained entrance into the cerebro-spinal fluid circulation. In the remaining case, (Eageton, Case VI), it probably was the removal of the inflammatory exudate from the sub-arachnoid spaces that allowed the already present meningeal reaction to complete the cure.

The remaining five of the twenty-one cases of local origin all began with an otitis; one reported by Gradenigo,[62] as previously noted, is not above the suspicion of being a skin contamination; one (Netter), is incompletely reported; in two (Day, and DuBois and Neal), the curative agent may be regarded as the intra-spinous

injection of a serum stimulating the protective meningeal reaction; and in one (O'Malley), cure followed a mastoid operation and lumbar puncture.

Probable Curative Agent:

Of the twenty-nine cases of recovery from a general suppurative meningitis, five of blood-stream—pneumococcic—origin (Litchfield), were treated by intra-spinous and intra-venous specific serum —Keyes'. Of the twenty-one of local origin, seventeen were operated and four unoperated (Gradenigo, Netter, Day, Case III, and DuBois and Neal). In three cases (Gobell's, Kummell's, and Tedesco's) the origin was not stated.

The operation apparently was the chief curative agent in the seventeen of the twenty-one cases of local origin (Kausch, Held and Kopetsky, Grossman, Beck, Bondy—three cases, Watson- Williams, Poirier, Alexander, Kostlivy, Gobell, Day—two cases. O'Malley, Scott, and Eagleton). Seven of the twenty-nine (Netter, Kummell, Tedesco, DuBois and Neal, Gradenigo, Day, and Lewkowicz) were not operated upon after the development of the meningitis, although the last three previously had been subjected to an operation.

In the seventeen cured by operation, the operation consisted of:

(a)—The removal of the intra-dural source of infection, combined with lumbar punctures—eight cases; three, intra-piarachnoid abscess (Held and Kopetsky, Poirier, and Kostlivy), and one, perforating dural wound (Kausch); two of brain abscess (Day), and one, of sinus thrombosis (Beck), were followed by drainage of the cerebro-spinal fluid system—Haines, operation; and one, compound fracture of the skull (Eagleton), by irrigation of the sub-arachnoid space alone.

(b)—Drainage of the labyrinth and incision of the dura, combined with lumbar punctures—one case (Alexander).

(c)—Drainage of the labyrinth combined with lumbar punctures —three cases (Grossman, Bondy, Case I, and Scott).

(d)—Drainage of the labyrinth, combined with lumbar punctures and intra-venous and the intra-thecal injections of serum—one case (Watson-Williams).

(e)—Removal of adjacent focus of infection (extra-dural abscess) and incision of dura, with lumbar punctures—one case (Bondy, Case II).

(f)—Drainage of adjacent focus of infection, with lumbar punctures—two cases (Bondy, Case III, and O'Malley, mastoid operation).

(g)—Laminectomy—one case (Gobell, unstated origin).

In the seven unoperated cases, apparently the chief curative agent was:

(a)—Lumbar punctures alone in four cases (Gradenigo, Kummell, Netter and Tedesco).

(b)—Lumbar punctures with intra-spinous injections of anti-streptococcus serum and vaccines in one case (Day).

(c)—Lumbar punctures with intra-thecal injection of meningococcus serum and intra-venous injection of anti-staphylococcus serum in one (Dubois and Neal).

(d)—Lumbar punctures and sub-cutaneous injections of staphylococcus vaccine prepared from the cerebro-spinal fluid in one (Lewkowicz).

Infecting Micro-organisms:

In the thirty-one recovered cases the infecting micro-organism was:

(a)—Pneumococcus in eight (Beck, one case, the two referred to by Dubois and Neal, and Litchfield, five cases).

(b)—Staphylococcus in six (Gobell, Kausch, Poirier, Kummell, Gradenigo, and Lewkowicz).

(c)—Bacillus Influenza in one, case of brain abscess (Day).

(d)—Streptococcus in ten (Tedesco, Netter, Dubois and Neal, Watson-Williams, Alexander, Day, serum treatment, and Day, brain abscess, Bondy, CaseII and Case III, and Scott).

(e)—Gram positive in one (Kostlivy).

(f)—Diplococcus in two (Grossman, and Bondy, Case III).

(g)—Diplococcus intercellular, extra-cellular, and mixed, in two (Held and Kopetsky, and O'Malley).

(h)—Diplococcus intercellular and extra-cellular in one (Eagleton, Case VI).

Note 1: *Typhoid Meningitis.* In a compilation by Baumgartner and Olsen[63] of all the cases of typhoid meningitis, no recovery followed.

Raymond F., and Siccard, J. A.:[64] Epidurite purulente lombaire a bacillus d'Eberth dans la convalesence d'une fievre typhoide. Paraplegie. Ponction lombaire; Bull. et Mem. Soc. Med. d. Hop. de Paris, 1905, Vol. XXII, p. 860.

Note 2: Tuberculous Meningitis. Hollis and Pardee[65] have collected 38 cases of verified tuberculous meningitis ending in recovery, to which they have added two of their own, making a total of 40 cases reported in the literature. The curative agents employed are fully reviewed. They advocate frequent spinal punctures and the intra-spinous injection of meningococcus serum, believing the latter curative, "(1)—by adding to the spinal fluid certain anti-bodies which it is unable to develop itself, and (2)—by the introduction of a foreign protein." An examination of their cases would suggest that it is the action of the foreign proteid in lighting up a protective meningitis, (as advocated in this study).

which is the chief curative agent, the cell count of the cerebro-spinal fluid increasing enormously after the injections.

DEDUCTIONS.

The following conclusions may be drawn from an analysis of the thirty-one collected cases of recovery from a general suppurative —non-meningococcic—meningitis.

I—The number of cures from general suppurative meningitis, either of local or blood-stream origin, is even smaller than generally is supposed. Consequently, general suppurative meningitis—with micro-organisms free in the cerebro-spinal fluid system—is an almost uniformly fatal disease.

II—The misconception that a fair proportion of recoveries of general suppurative meningitis of local origin have followed from operation, has originated from a failure to appreciate the diagnostic and pathological distinction between a lumbar puncture which reveals a cloudy fluid filled with leucocytes but without bacteria—a protective and reparative process—and the puncture which yields a turbid or a clear fluid containing micro-organisms—a general suppurative meningitis. Consequently a considerable numebr of the reported cures of suppurative meningitis were in reality cases of protective meningitis, and while clinically they presented severe meninegal symptoms, the general cerebro-spinal fluid system was not invaded by bacteria.

III—In the reported cases of cure from meningitis of blood-stream origin—five, of pneumococcus meningitis, (Litchfield)—the successful treatment undoubtedly was the intra-spinous injection of a serum which controlled the bacterial meningeal invasion, and probably stimulated the protective meningeal mechanism. The elimination of the blood-stream infection—either by the action of the blood itself, or with the assistance of a serum or vaccine acting upon the circulating blood—plays but a secondary part. In proof of this witness the immense number of cases of pneumococcic blood-stream infection, which recover spontaneously, contrasted with the almost uniformily fatal result when the meninges become bacterially involved. There is, however, microscopic evidence to warrant the belief that 25 per cent of all general pneumococcic invasion are associated with a protective meningeal reaction.[66]

IV—Of the twenty-one reported cures from general suppurative meningitis of local origin, over two-thirds, or from fifteen to seventeen cases, had had prior to the general meningeal infection a meningeal protective reaction which was in action at the time of the general meningeal invasion. The invasion of the cerebro-spinal

circulatory system in these cases possibly was a temporary breaking loose of micro-organisms through the limiting and protective process.

V—Consequently, on account of this relatively large proportion of cures in all the cases of local origin, it is reasonable to assume that it was largely through the presence and immediate action of this protective process that recovery resulted; assisted by the evacuation of the causative localized, intra-dural suppuration, (*i. e.*, brain abscess, intra-piarachnoid abscess, or a suppurative labyrinthitis), and in rare cases possibly by the elimination of a causative extra-dural suppurative focus, which may be associated with a pachymeningitis interna—such as extra-dural abscess, sinus thrombosis or adjacent bony caries.

If this deduction be true it would suggest that it is of proime importance in the treatment of general suppurative meningitis that the surgeon direct his efforts to stimulating and assiting the protective meningeal mechanism. In the absence of a specific serum such as is now available for meningococcus meningitis this can best be accomplished by keeping open the cerebro-spinal pathways by sub-arachnoid irrigation.

VI—Of the small number of recovered cases of local origin in which no protective process apparently was in operation—five in all—the curative agents were so diversified that they may be regarded as more or less accidental.

VII—Lumbar puncture alone undoubtedly is of therapeutic value. Probably because it is the means of removing some of the infected fluid, and possibly, by keeping open the cerebro-spinal pathways; it relieves, temporarily, the cerebral compression which favors intra-cerebral, and probably meningeal suppuration.

VIII—The number of cures in which an intrathecal injection of a serum has been employed—either alone, or in conjunction with other measures—is too large to be ignored. Consequently, it would suggest that the intra-spinous, intra-ventricular or cerebro-sub-arachnoid injection of a serum even if it possesses no specific action on the particular type of infecting micro-organism, undoubtedly has a therapeutic value, probably by stimulating a protective meningeal reaction—the well recognized tissue reaction to a foreign proteid.[67]

CONCLUSIONS.

From the foregoing analysis it would appear evident that, given bacteria free in the cerebro-spinal fluid circulatory system, (in the presence of a localized focus of suppuration within or involving the dura), drainage of the intra-dural focus of infection, associated with lumbar puncture, offers a slight hope of recovery. In the ab-

sence of such an intra-dural suppuration, stimulation by a serum injected into the spinal or cerebral spaces of the cerebro-spinal system offers a slight prospect of recovery. But logically, in all cases sub-arachnoid lavage holds out a prospect not offered by any other therapeutic agent, as lavage alone keeps open the cerebro-spinal system pathways.

Whether this hope will be fulfilled in the future, experience alone will decide.

REPORT OF THREE CASES OF SUBARACHNOID IRRIGATION WITH ONE RECOVERY.

Case VI. General Suppurative Intra and Extracellular Diplococcus Meningitis Following Compound Fracture of Skull, Treated by Intra-Cerebral Subarachnoid Irrigation; Recovery.

Resume of Case: Compound fracture of base of skull from which patient apparently was recovering, but continued to complain of constant and severe headache. On the fifteenth day after the injury, severe meningeal symptoms. Lumbar puncture revealed diplococci, both intra and extra-cellular. Intra-spinous injection of meningococcic serum. Sub-arachnoid irrigation promptly followed by recovery.

Patient admitted to Newark (N. J.) City Hospital, March 28, at 6:45 p.m.

Clinical Diagnosis: Compound fracture of skull.

Notes of Receiving Room on Admission: Struck by trolley car; patient unconscious and in shock; had a large, stellate laceration of scalp; bleeding from mouth, nose and left ear; right pupil enlarged, reacted slowly to light; external strabismus of that eye; abrasion of left elbow; no other visible injuries; may have had fractured ribs on left side, but not definitely made out.

Physical Examination by House Surgeon: General appearance—well developed, well nourished, white female of 45, not conscious, pale, cold, clammy, aroused at times.

Head: Laceration of scalp 5 inches long running anterior from post-parietal region to temporal region, and another laceration running perpendicular to it at its middle and below it.

Eyes: Pupils equal, regular, reacted to light; sclera clear; conjunctiva not injected; external strabismus of both eyes; no nystagmus; eye grounds—discs pink, edges hazy.

Nose: Dried blood in both nares.

Ears: Right, negative; left, fresh blood in auditory canals.

Mouth: Blood in mouth; patient spat up dark brown blood. Jaws held rigidly so that examination of mouth was impossible.

Neck: No rigidity; no injury detected.

Chest: Breasts negative; heart, rate slow, of good quality, no murmurs; lungs negative; ribs negative.

Abdomen: Slightly pendulous, adipose, no masses.

Extremities: Negative.

Reflexes: Knee and ankle jerks not elicited; no clonus; no Babinski; no abdominals; no Oppenheim.

Nodes: Not palpable.

Skin: Negative.

Provisional Diagnosis: (1) Fracture of left base of skull involving middle fossa and parietal bone; (2) Laceration of scalp.

Antecedent Personal History: General health good; no injuries or shocks. Patient always had "stomach trouble." Father died from accident at age of forty; mother died from rheumatism at sixty-five.

March 28: (*Nurse's Notes*): "Patient admitted to ward; semi-conscious; color poor; pulse fair; camphor and ether mixture given in R. R. Lay quietly. Blood pressure taken. Vomited bloody fluid. At 1 a. m. delirious and very noisy."

Roentgen Diagnosis: Right negative; linear fracture left temporal parietal region.

For a number of days patient vomited at times greater or less quantities of bloody fluid; rational at intervals only; noisy and talkative at intervals. Spinal tap; fluid obtained under pressure; 13 cc. of fluid removed. General condition remained unchanged, except for slight improvement in pulse (76). Blood pressure—100-130 Systole; 60-80 Diastole.

March 29: Eye grounds negative.

March 30: Dressing on head pulled off by patient. Clean dressing applied by nurse. Wound looked as if it might be infected. Patient restless and excited. Urine negative. Eye grounds negative. Blood in left ear. Temperature 99-100°

April 1: Eye grounds negative. Patient spent a fairly good night. In the following days head ached at times, and patient complained of pain around heart.

April 5: Patient insisted upon taking bandage from head.

April 6: Fairly comfortable by day; slept at short intervals at night. Temperature normal. Pulse 76. Blood pressure 120-80.

April 8: Somewhat brighter, but still had headache.

April 9: Patient still complained of headache; otherwise condition about the same as before. Following days severe headaches.

April 13: Right disc outline very slightly hazy; vessels full and disc slightly swollen. Lumbar puncture performed and 20 cc. clear fluid obtained without pressure. Patient more comfortable and headache less severe.

April 14: Patient restless and fretful.

April 15: Patient still restless, trying to get out of bed. At 4:30 a. m. got out of bed, fell and struck her head. In shocked semi-conscious condition. Pulse of good quality. Patient very restless. At 7 a. m. unable to rouse patient. (Patient had acted as if she had a slight convulsion on the floor when she fell.) Blood pressure irregular—Systole 118-95; Diastole 70-55. Temperature normal. Pulse 72. Respiration 20. At 8:45 a. m. patient in a stuporous condition from which she could not be roused. Pupils equal, reacted to light; right disc indistinct. Knee jerks active. No Oppenheim. At 5:30 p. m. a lumbar puncture was performed; 20 cc. cloudy fluid obtained under slight pressure. Pathological report on cerebro-spinal fluid; intra and extra-cellular diplococcus. "Looks like meningococci infection" patient semi-conscious. Applied ice cap to head.

April 16: Voided involuntarily. Complained of headache. Temperature and pulse normal. Neck rigid. At 3:30 p. m. lumbar puncture performed; 32 cc. cloudy fluid obtained under slight pressure. Twenty cc. anti-meningococcic serum injected.

Operation at 7:30 P. M. Craniotomy and irrigation of sub-arachnoid space.

Procedure: Exploration along line of fracture showed probable extension into base over temporal region. Closure and drain. Trephine opening in left frontal region in hairline three-fourths of an inch from medial line; cerebello-medullary cisterna entered by occipito-atlantal puncture; at a depth of about three and one-quarter cm. cloudy fluid was obtained. Irrigation with physiologically balanced modified Ringer's solution at body temperature between openings through sub-arachnoid space of about 200 cc. Toward end of irrigation the recovered fluid became bloody and then ceased to flow. Dura in frontal region distended, and on removal of needle from frontal opening, fluid followed:

evidently a block had developed, as fluid was being recovered a short time previously at the rate of about sixty drops per minute. The block may have been due to injury to a pial vessel.

Examination of fluid showed it to be cloudy, yellowish gray. Pus +. Chemical globulin, large amount. *Cytogeny*, polynuclears +. Bacteria meningococcus +.

Immediately after operation the patient was delirious and very noisy, but condition gradually improved.

At 11 p. m. patient out of ether; color and pulse good quality; pulse rate 120.

April 17: At 2 a. m. patient delirious and noisy. One-sixth of a grain of morphine and one one-hundred-and-fiftieth of a grain of atropine given, after which patient slept the remainder of the night. At 4 a. m. temperature was 101°; pulse, 88; respiration, 22. At 7 a. m. her general condition apparently was excellent. Patient conscious, and so remained. At 8 a. m. lumbar puncture was performed; 30 cc. *slightly* cloudy fluid obtained under considerable pressure. Twenty cc. anti-meningococcic serum given. Patient had a comfortable afternoon that day. At 8:30 p. m. lumbar puncture gave 15 cc. slightly cloudy fluid under slight pressure. Twenty cc. anti-meningococcic serum given. Temperature 101°; pulse 90.

April 18: General condition favorable; patient had slept at intervals through the night. Examination of fluid showed the color, yellow; transparency, cloudy; nebula +; globulin, large amount; polynuclears +; bacteria, smears none. At 8 a. m. temperature 99.2°; pulse, 88. At 9 a. m. lumbar puncture gave 30 cc. almost clear fluid under pressure. Twenty cc. anti-meningococcic serum given. Neck still rigid; knee jerks very sluggish. Kernig and Babinski in right side. At 10 a. m. lumbar puncture gave 25 cc. yellow, cloudy fluid under pressure. Fifteen cc. anti-meningococcic serum given.

April 19: Patient had slept all night and said she felt fine in the morning. At 9 a. m. lumbar puncture gave 32 cc. slightly cloudy fluid under slight pressure. Twenty cc. anti-meningococcic serum was given. Pathological report; fluid yellowish in color, cloudy; globulin, large amount; smears, no bacteria. Temperature, 100°; pulse, 90-96.

April 20: Patient slept all night, but voided involuntarily during the night. Temperature normal. Patient apparently entirely normal.

April 27: Discs clear. Patient insisted upon leaving hospital, regarding herself as well, not having had headache since the operation.

Case VII. Cerebral and Cerebro-Spinal Sub-Arachnoid Irrigation for General Suppurative, Meningitis (Streptococcic, Hemolytic) Originated from a Latent and Unsuspected Cerebral Abscess from a Gunshot Wound Inflicted Eighteen Months Previously. T. P.; male.

Resume of Case: About one and one-half years previously the patient attempted suicide by shooting himself in the right temple with a revolver. He was brought to the hospital and operated upon by me. I removed a pistol bullet from the left parietal region, the ball having passed through both frontal lobes, where there was a bursting fracture of the skull. The X-ray after the operation showed small fragments of bone or bullet near the wound of entrance, but the patient made a very good recovery and apparently has been quite well since. (It is interesting to note, however, that after this time his history states that he became a drug addict.) There was a recent neurological examination before his second admission to the hospital.

On June 7, a few days before his death, the patient was found by the police in a more or less dazed condition and brought to the City Hospital, where he was placed in the observation ward for psychopathics, apparently little attention being paid to his former cerebral injury. It was noticed that he had a stiff neck, the patient walking around the

ward in this condition. The intern in charge of the service performed a lumbar puncture which showed a cloudy fluid under pressure; when examined it showed numerous pus cells and many Gram positive cocci, many arranged in chains. A diagnosis of streptococcic suppurative meningitis was made, and the patient was transferred to my service.

Two attempts were made to wash out the sub-arachnoid cerebral space, and anti-meningococcic serum was given intra-spinously, with the idea of the serum stimulating an aseptic protective meningitis. The patient grew worse after the second operaton, but had appeared in much better condition after the first. Culture of the last fluid, shortly before death, showed definite hemolytic streptocci. Post mortem disclosed three latent abscesses, the oldest of which doubtless was present from the time of the original injury. One of them had perforated the lateral ventricle and slowly discharged its contents into it. From this the infection had passed to the basal cisterna and the cerebellar surface, the sub-arachnoid spaces of the cortex remaining unaffected.

History in detail: June 7, admitted to City Hospital at 10:20 p. m. Temperature 100.2° and pulse 100.

Examination, June 8: Poorly developed and purely nourished male adult, about 50 years of age, lying quietly in bed, acutely ill.

Head: Healed, irregular, depressed scar over right temporal region; bony depression just above the external occipital protuberance; healed, depressed, irregular scar over left superior temporal region; lividness, and some abrasion present over right eye.

Eyes: Rotary nystagmus present in right eye; less marked in left eye; moderate sub-conjunctival injection of right eye-ball; pupils round, regular, right } left; react normally to light and accommodation.

Nose: Abrasion on the bridge; tip turns toward the right; septum deviated to the left.

Ears: Present no sign of injury.

Mouth: Teeth in poor condition; tongue coated, fissured and furred; protrudes in the median line.

Neck: Marked retraction and rigidity.

Thorax: Normal contour; poor expansion.

Lungs: Resonant and clear throughout as far as can be ascertained with the poor co-operatin of the patient.

Heart: Borders slightly enlarged to the left of nipple line; first sound at apex replaced by a soft, blowing, inconstant murmur at base

Arteries: Moderate degree of sclerosis and thickening.

Abdomen: Liver one finger breadth below costal margin; no other masses or points of tenderness made out. Tache cerebrale is positive.

Genitals: Incontinuence of urine.

Reflexes: Deep reflexes present; hypo-active, but equal; double Babinski and double Kernig present; no ankle clonus obtainable.

Diagnosis: Streptococcus meningitis.

Physchiatric Examination, Wednesday, June 8: "I've been here three or four weeks." (Was admitted yesterday). Does not know day, month or year. Says "1821" At 10 a. m. says it is about 3 o'clock. "I dont know why I am here. I don't know who brought me here, either. I wasn't drunk. I don't know what was the matter with me."

Why do you stay here? "I dont know that either." Using drugs? "No." Anything hurt you? "No." Were you here before? "I imagine so." Have any trouble? We had quite an argument—my brother—a little family affair." Did the police bring you in? "I don't know." Did you try to kill yourself? "No."

"Harding is President; Wilson before him; Hauseling is Mayor."

"4X4=16+10=26—8=—well—" (then no answer).

"9X9=81+9=81—oh, well—" (Then no answer.)

"6X6=36+8= —well—" (Then no answer).

"20—1=19." (Correct, but slow).

"Before July comes August; before December, November; before October, September—" (after some thought).

Name the months of the year. "This month—" (Then no answer).

Memory for numbers poor—"3, 4, 8, 7." No dysarthia.

Both pupils very sluggish to light—both irregular. Suspicion of Romberg. Knee jerks exaggerated. Urine dribbled during examination. Executes commands sluggishly. Gait somewhat spastic. Marked tremor of fingers.

June 9: Temperature 99.2°, pulse, 110. Incontinent; neck stiff.

Lumbar puncture: Fluid cloudy under slight pressure; Wasserman negative; direct smears show streptococci. At 8 p. m. pulse 150-90.

Operation at 11:30 p. m.: Cerebral and spinal sub-arachnoid irrigation. Three trephined openings made in right fronto-parietal region; in the first it was found impossible to enter fluid because of apparent adhesion of dura to brain, although the opening was away from the skull opening through which the ball had been extracted, by at least one and one-half inches. Two other openings were then made: opened into the sub-arachnoid space, and tap made into cisterna magna. Needle inserted underneath dura and arachnoid and surface of brain flushed out with Ringer's solution and drained through needle in cisterna. Fluid at first yellow, turbid, then bloody, then yellow again. Process repeated twice through trepine one inch posterior to first opening on the same side. Later the excess fluids were drained through a lumbar puncture. Flaps closed with black silk.

12:45, midnight: Pulse 130—good quality; color fair.

2:40: Out of ether.

June 10, 8 a. m.: Pulse 96; "good and regular." Temperature 101°. Patient lies quietly, seemingly staring into space. Blood pressure 120-90.

12 Noon: Temperature 102°; pulse 120. Lumbar puncture; fluid cloudy, slightly blood-tinged; anti-meningococcic serum injected intraspinously.

6:30 p. m. Nurse's Note: "Seems to understand when spoken to. Does not talk himself. Patient incontinent." Laboratory report: "Fluid contains pus and blood; Gram positive cocci."

4 p. m. Temperature 100°; blood pressure 140-95.

June 11, 8 a. m.: Temperature 99°; pulse 96. Nurse's Note: "Patient appears to be much better this morning. Answers intelligently to questions. Neck still very rigid and head thrown back.

12 Noon: Lumbar puncture done; 28 cc. withdrawn, fluid cloudy, slightly yellow, under slight pressure. Anti-meningococcic serum 20 cc. injected. At this time I examined the patient; the man was in excellent condition. He recognized me at once and recalled all about the previous experiences in the hospital eighteen months ago.

3 p. m.: Lumbar puncture; fluid cloudy, under moderate pressure; 30 cc. serum injected.

4:45 p. m.: Blood pressure 140-95.

6 p. m.: Second sub-arachnoid irrigation. Anterior flap opened; stitches removed; needle inserted under arachnoid and irrigation carried on as on the previous day. Drained through cisterna and lumbar puncture. Fluid yellow and somewhat turbid. Flap closed with black silk.

7:15 p. m.: Patient returned to ward. Pulse 130, of good quality.

10 p. m.: Pulse 120.

11:30 p. m.: Blood pressure 170-130.

June 12, 4 a. m.: Blood pressure 155-125. Pulse 120.

8 a. m.: Patient's condition did not seem so good. Pulse fair. Answered when spoken to.

8:30 a. m.: Patient suddenly became cyanosed and pulseless and ceased to breathe.

AUTOPSY AND REPORT BY DR. HARRISON S. MARTLAND.

External Examination:

"Showed a male body, about fifty years of age. There is slight recent loss of weight. Moderate rigor in smaller and larger joints and moderate lividity over dependent portions of body. The skin over the entire body is free and clear.

Examination of the head shows a recently shaved head, with a recent sutured operative incision over the right temporo-parietal region, the surrounding skin being covered with silver foil.

Over the right temporal region is an old, small depression and opening in bone, over which the skin is firmly adherent and contains a small, old scar wound of entrance of bullet.

Over the left parietal region is an old linear scar under which the bone is replaced by membranous tissue for a distance of about 3 cm.—the wound of exit, or surgical extraction of bullet."

Internal Examination:

Head: "The old wound of entrance of bullet situated in the right temporal region shows an old scalp scar, adherent overlying skin, and a small opening in the bone with rounded edges; the dura beneath is firmly adherent and there are numerous adhesions between the dura and pia-arachnoid about this small area. In the cortex of the middle of the F. I. lobe of the right frontal lobe is a small, cortical and sub-cortical abscess, measuring about 1.5 cm. in size and filled with thick, creamy pus containing a few small fragments of bone; the wall of the cavity is about 2 mm. in thickness.

Situated deeper in the brain substance, its outer edge encroaching on the small abscess already described, is another more recent and larger abscess, measuring about 2.5 cm. in diameter,and containing a slightly greenish pus; its walls are about 1 mm. in thickness. The lower and inner portion of this abscess cavity communicates with a smaller and still more recent abscess cavity, about 1.5 cm. in size, which leads directly into the anterior horn of the right lateral ventricle, at the opening being soft and necrotic brain tissue.

The right ventricle is distended with greenish, yellowish pus, and there is an extensive exudate clinging to the choroid plexus; the ependyma lining the cavity contains numerous punctuate hemorrhages, and the surrounding brain tissue is soft, oedematous, and in places distinctly necrotic. This same condition is found in the left lateral ventricle, although not to so marked an extent.

The third ventricle contains purulent exudate and fluid pus, and the interpeduncular cisterna is distended with purulent exudate. The fourth ventricle is dilated, as is also the iter, and contains a purulent exudate and free pus. This has leaked out through the foramina of Magendie and Luschka and infected the cisterna magna, which is filled with a plastic, purulent, yellowish exudate, which extends up over the inferior and lateral surfaces of the cerebellum and stops. The cisterna basilis and chiasmatis contain considerable plastic exudate, which apparently comes from extension from the cisterna magna, and possibly also from the close proximity of the infected third ventricle. The outer surfaces of the brain and the spinal cord and its meninges are practically free from exudate.

Over the right cerebral hemisphere in the calvarium there have been three recent trephine openings, all about 2 cm. in size, and filled with recent blood clot. The dura under each has been sutured and the anterior opening over the right frontal lobes shows a small hemorrhagic extravasation in the leptomeninges—a needle puncture for washing out the sub-arachnoid space. The other two show a similar but not so marked a condition.

Extending from the wound of entrance in the middle of the frontal lobe passing across the frontal lobe on its superior surface, to the left and slightly backward, through the left frontal lobe on its superior surface, and stopping at the bone opening in the left parietal region, is

a somewhat collapsed brain tract of the old bullet wound. The brain tissue is stained a gumboge yellow around this old tract and there are numerous piarachnoid adhesions over this area. The anterior trephine opening for washing the sub-arachnoid space is included in this area, and the surrounding adhesions probably prevented the free admittance of fluid.

The base of the skull and accessory sinuses are free and clear. In the posterior part of the foramen magnum there are two small puncture wounds through the dura and arachnoid in the space between the skull and axis. There is a small amount of recent blood about these openings. The anterior surface of the pons and medulla shows considerable recent hemorrhagic extravasation in the cisterna pontis, but there seems to be no anatomical connection with the needle punctures, and it probably is due to toxic action of streptococcus in the exudate in this region.

The spinal cord is practically free and clear.

Pertoneal Cavity: Viscerna in normal position; free and clear.

Pleural Cavities: Free and clear.

Heart: Heart muscle soft, friable, and pale red in color, with indistinct bundles; otherwise free and clear.

Lungs: Show terminal oedema and small areas of terminal broncho-pneumonia.

Examination of other viscera is negative except for cloudy swelling of heart muscle, liver and kidneys.

Probable Cause of Death: Gunshot wound in head, penetrating, wound of entrance in right temporal region, crossing both frontal lobes supraventricular and stopping in a bursting fracture of the left parietal bone. Wound made over one and one-half years ago in attempted suicide.

Secondary or Terminal Lesions:

1. Encapsulated abscess of right frontal lobe, cortical; recent progression of infection from encapsulated abscess to brain substance around abscess, with formation of two more recent abscesses, one finally rupturing into the anterior horn of the right lateral ventrical.

2. Spreading, generalized suppurative ependymitis, extending rapidly throughout the entire ventricular system.

3. Terminal suppurative meningitis, fairly well limited to the cisterna magna, cisterna pontalis and cisterna basalis.

4. Three recent operative trephine openings with small puncture wounds of the underlying piarachnoid and sub-occipital needle taps for washing the sub-arachnoid space.

5. Cloudy swelling heart muscle, liver and kidneys and terminal broncho-pneumonia.

Historical Landmarks: Old bullet tract through both frontal lobes.

Autopsy Findings: Old gunshot wound of superior frontal lobe, with dura adherent over area of exit of bullet, and underneath an abscess (small) with organized wall communicating with larger abscess, resulting in basal meningitis and extending from cisterna—filled with pus—up to lateral ventricles and producing an ependymitis and encephalitis.

Comments: Of course, a brain abscess should have been suspected, and an attempt made to drain it, possibly associated with sub-arachnoid irrigation. Sub-arachnoid irrigation, however, would, of course, have been useless without the removal of the cause.

In view of the excellent condition of the patient just prior to the second irrigation—and the subsequent rapid rise of blood pressure and the sudden termination, one is forced to the conclusion that death resulted from cerebral compression, probably induced by the irrigation. In addition to the over-looked abscess, the patient probably was over-treated both as to lumbar puncture and serum injections, and sub-arachnoid irrigation.

Case VIII. Case of Delayed Pneumococcus Meningitis from Cititis. Incomplete Subarachnoid, Spinal and Cerebral Irrigation on Tenth Day of Disease; Child, 8 years of age. Seen with Dr. Blackhorn.

History: Two weeks previously patient had an earache; paracentesis was made by Dr. Barkhorn, followed four days later by a mastoid operation. Large antrum found. For the next week nothing unusual noted, except that on second day child vomited several times. It then appeared perfectly well, and was discharged from hospital.

One week after vomiting attack child awakened in the middle of the night with sudden and severe pain in the head and again vomited. The vomiting continued about every hour for eighteen hours, and then stopped. During the next three days, it had continuously high temperature running from 102° to 105°.

When seen in consultation, patient had stiff neck, coma, cell count of cerebro-spinal fluid over 900, fluid under pressure, globulin increased, Fehling's reduced.

Operation, April 8: (Two weeks after initial earache and ten days after first vomiting attack.

Trephine hole drilled in right frontal region. Mastoid of right side then widely opened, both above and behind; nothing revealed. Dura punctured over the tegmen; small amount of cerebro-spinal fluid; no hemorrhage. A lumbar puncture was then performed and sub-arachnoid drainage established. Drew away about 100 cc. of turbid fluid, allowing about 55 cc. of fluid to run into sub-arachnoid space of frontal region. The tension in the lumbar puncture needle rapidly fell. So far as we could make out the irrigation was not very successful. Could not tell whether the out-flow was increased by the fluid which entered from above or not, although the out-flow contained cloudy fluid with considerable exudate. Gradually the out-flow fluid stopped running.

The injected fluid was introduced very slowly, at about the rate at which cerebro-spinal fluid normally drops from a lumbar puncture needle. The temperature of the in-flowing fluid was kept continuously at the body temperature. At the completion of the operation the child's pulse began to rise a little; otherwise there was no apparent effect constitutionally. Three hours after irrigation, temperature was 101°, and pulse 108.

Laboratory Report of Fluid: "Cloudy, globulin increased, Fehling's slightly reduced. Smears show numerous streptococcus mucosus—sometimes known as pulmococcus mucosus, or pneumococcus Type III. Blood culture negative."

Cadaver Experimentations. Following this failure I demonstrated on a cadaver that with an in-take needle in the frontal region and the out-flow needle in the lumbar region, on injecting the upper needle, only thirty drops per minute could be recovered from the spinal region. However, with the out-flow needle in the occipito-atlantal region, fluid could be irrigated from the frontal region, the fluid being recovered freely in a continuous stream from the occipito-atlantal needle. Also, that using the occipito-atlantal needle for the in-take, fluid could freely be recovered from the lumbar region.

April 9: Since operation the child's condition apparently has greatly improved. The temperature has continued high, however,—around 103°—but the pulse since last night has become slow—88.

April 10: The child presents the usual picture of meningitis—at times restless and again quiet; temperature 102-104°, but pulse slow—68-92.

Lumbar puncture; Laboratory report: "Fluid cloudy, gobulin increased, Fehling's reduced very little. Smear streptococcus mucosus, number only slightly increased over previous specimen."

April 11: Several involuntary bowel and bladder movements. Sleeps at short intervals. Delirious at times. Temperature 104°; pulse fast —135.

April 12: Patient weaker than yesterday. Takes little nourishment, but swallows well. Delirious and restless. Temperature 103-104.4°; pulse 126-130.

Second Irrigation, from lateral cisterna to lumbar region. With the experienced data I had obtained, I made a puncture through the lower portion of the mastoid wound, over the posterior fossa, a small opening being made in the dura, and the inflow needle placed in the lateral basal cisterna, and an attempt made to irrigate from this through to the lumbar region. The fluid obtained by the lumbar puncture was at first almost pure pus. This operation also was not very successful because of the large amount of exudate, which continually clogged the needle in the lumbar region and necessitated the frequent passage of a stilette to free its lumen. The amount of fluid washed through was not more than 55 cc. At the end of this procedure the child appeared to suffer slightly from shock; the pulse began to rise and there was a tendency to some excessive moisture of the skin, doubtless because of the prolonged manipulation. There was no respiratory involvement whatever. This operation, also, I regard as more or less of a failure, although a large amount of exudate undoubtedly was washed away.

Laboratory report of fluids: "Fluid recovered very cloudy; pus organisms increased—double that of April 10; Fehling's not reduced; globulin greatly increased."

Following the irrigation the child apparently was much improved. Temperature 103-104°; pulse 130.

April 13: Nurse's notes during night following second irrigation: "2 a. m. Patient seems to rest quietly the last few hours; 4 a. m. Patient resing quietly; seems to be improving. Temperature still high; pulse slower."

Twenty-four hours after the second irrigation, at the request of the child's family, who had noted a marked improvement after each of the previous irrigations, another irrigation was done.

Third irrigation, 5 p. m. Occipito-atlantal puncture, reaching the basal cisterna at 4¼ cm., the landmarks given by Ayer being followed. The dura was distinctly felt by the needle, and on perforating it a large amount of very cloudy fluid was recovered. The inflow needle was then introduced through the old trephine opening in the frontal region and I was enabled to wash through a large amount of fluid, although the needle in the medullo-cerebellar cisterna was constantly being clogged by the exudate. At first the flow was free from the out-flow needle, but gradually it became much thicker. At this irrigation at least 200 cc. was washed through.

Laboratory report of fluid: "Pus and organisms greatly increased. Streptococcus mucosus."

Following this third irrigation, the pulse was fast—168; respiration also much faster—40; temperature 102-104°.

April 14. Notes: "Left internal strabismus. Convulsive movements inward of left eye, the right remaining fixed, looking sharp ahead. Both O. E. veins full, not tortuous. Arteries also full. Right disc may have slight indistinctness of upper margin." The pulse has been faster since the last irrigation. Exudis twenty-four hours after last irrigation.

AUTOPSY AND REPORT, BY DR. FREDERICK SUTTON.

"Simple extensive mastoid wound, right side. Moderate hemorrhage under scalp. Half inch frontal trephine opening. Extra-dural blood clot—small. On opening dura, moderate sub-dural hemorrhage—right side—extending back to tentorium. Two clean needle punctures in dura. Needle puncture in dura atlanto-occipital joint slightly to right of center. Slight extra-dural exudate about mastoid wound. Needle opening just above external semi-circular canal. Petechial hemorrhage in dura over right frontal area, similar condition sub-dural.

Brain: Superior surface microscopically clear of pus. Considerable oedema and congestion. Ruptured blood vessel beneath needle puncture,

small amount of hemorrhage. Slight area of necrosis corresponding to needle opening in mastoid wound. Moderate pus in both Sylvian fissures, bathing pons and cerebellum down to cord. *More marked on left side than on right.* Right tri-facial nerve oedematous, and containing pus infiltration (5th). Apex of petrous bone not involved. Right lateral sinus patent; no clot. Right superior petrosal clear. Left side lateral sinus branches one and one-quarter inch from torcular, lower entering one-half inch below the upper. Both branches patent. Left superior petrosal patent. All sinuses clear. No diseased bone found in mastoid. Optic nerves clear. Sella and pituitary clear. Pus in left post-ethmoids. Smear showed streptococcus mucosus. Right ethmoid clear. Muco-pus in right spheonid—smear showed streptococcus mucosus. Left sphenoid free and clear. Both frontal sinuses dry. Right semi-circular canal clear. Left middle ear—smear—numerous streptococcus mucous."

Comments: The meningeal involvement probably occurred in the second day after the mastoid operation, but was localized and controlled by the protective meningeal mechanism until one week later, when it became general.

From my experience of this case it would seem that it is a dangerous thing to keep on irrigating after the flow stops, because undoubtedly at the completion of this operation it caused compression symptoms. The child was more or less comatose at the time of the irrigation. The important thing to remember is that it is feasible to wash directly across the brain, over the basal cisterna, into the occipito-atlantal space; but we must not wait until the exudate is firm, because the exudate fills the needle. Also, we must use a graduated needle; and the needle must not be too long, as was this case, because the longer the needle the more apt it is to become filled with the exudate.

The post-mortem undoubtedly showed that the side irrigated was very much less infected than the opposite side. It also showed how easy it is to alter the needle to slip under the dura into the arachnoid space and cause a hemmorhage, unless it is held very firmly. The needle that goes into the sub-arachnoid space should be guarded. It is not necessary to guard it in the occipital-atlantal space.

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"The fact that the same numbers of virulent streptococci on injection both into the sub-arachnoid space and into the peritoneal cavity result in a fatal infection indicates that the sensitivity of the meninges was not alone the determining factor in the reaction. It seems most likely that in both cases we are dealing with a mesothelium-lined sac serving as a good incubator, with enough nutriment for the development of the bacteria injected and with very slight inhibitory power of the body elements." However, "there are immune bodies in the cerebro-spinal fluid of immunized animals." FELTON, L. D.: The Intra-Meningeal Virulence of Micro-Organisms; *Monographs of the Rockefeller Institute for Medical Research*, March 25, 1920, No. 12, p. 45.

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DEFICIENCY DISEASES OF THE EAR, NOSE AND THROAT: 1. OTOSCLEROSIS. 2. HYPERPLASTIC ETHMOIDITIS. A PRELIMINARY REPORT.*

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Attention today is centered on the role of vitamines in relation to specific disease entities, to various obscure pathological states in both man and animals, and to various ill defined functional disorders. Almost every special field in medicine has offered some phase of the possible tissue damages or functional disablement by certain deficiencies in diet. The clinician and the laboratory worker have proven that scurvy and berri-berri are caused in man by the consumption over a long period of time of diets which are deficient in the necessary vitamines.[1] They have established that these diseases can be prevented by the addition to the dietary of vitamine containing articles, and if present can be benefited to a great extent by furnishing the deficient factors. The pediatrician has proven that rickets, a disease of early childhood, has for perhaps its most important etiological factor another vitamine deficiency and that the addition of cod-liver oil to the dietary will cure the disease because of its Fat Soluble Vitamine Content[2 3] The obstetrician has offered sterility, amenorrhea, inability of women to suckle their children—all related to nutritional disorders.[4 5] In the field of ophthalmology—a condition known as xerophthalmia can be produced by the feeding of animals (rats) food from which the fat soluble accessory is absent, the bacterial invasion playing a secondary part.[6 7] Dental caries, pellegra, and war edema have likewise been proven clinically and experimentally to be the result of deficiencies in the diet.[8 9] No phase of this subject has, up to this time, been touched upon in the field of rhinology or otology, and does it seem likely that these parts can escape when such definite changes in every tissue and organ of the body—bone, blood vessels, nerves, and so on, can be produced experimentally and observed clinically by omitting from the diet certain ill defined substances which are needed in but minute proportions but yet are essential to life and the proper functioning of the body cells. Yet in animals, it has long been recognized that the "snuf-

*From the Clinic of Dr. Joseph C. Beck and Dr. Harry L. Pollock, North Chicago Hospital.

fles" of hogs, is primarily a nutritional disorder—of the "nutritional infectious" nature; and pediatricians have called attention to the frequent coincidence of nasal diphtheria and latent or sub-acute scurvy.[2] What part then does deficiency disease play in producing, if at all, any pathological state or malfunctioning of parts in the ear, nose or throat, other than fertile soil for the growth of saprophytes or pathogenic organisms? Can certain nasal or aural conditions be considered but a local manifestation of some general constitutional disturbance, some nutritional disorder, for instance, or may not particular tissues show a low resistance to particular vitamine deficiencies, tissues either least essential to life or more highly specialized in their function? With this in mind, we have made an effort during the past year of observing the possible relation of dietary deficiencies to various nasal and aural conditions which were particularly of more or less unknown etiology and which pathologically showed changes not of an inflammatory nature nor directly due to bacterial invasion.

Of these oto-sclerosis and hyperplastic ethmoiditis appeared to be the most conspicuous, and our observations are the purport of this preliminary report.

Before going further it would be well to state more clearly the modern conception of a deficiency disease.[9] A diet to maintain proper nutrition must be proper quantitatively and qualitatively; thus it must have a sufficient caloric value and must contain sufficient constituents as

1. Mineral salts, (including iodin).
2. Carbohydrates.
3. Fats.
4. Proteins (sufficient in amount and in type *i. e.*, amino acids).
5. Vitamines.

Any disproportion or deficiency will produce defective nutrition. It can readily be seen how our modern diet with its sterilization and purification, its cooking and canning of food-stuffs, may well be deficient particularly in its vitamine content.

Oto-sclerosis: The basis for even the assumption that oto-sclerosis may be a manifestation of a deficiency disease was obtained from the analogy of the pathology in rickets and in this condition, so much that I have called it a latent "adult rickets."

According to Seibenmann, its pathology is essentially a primary softening or spongification of the bony capsule of the labyrinth, especially in the vicinity of the foot-plate of the stapes and

larger coils of the cochlea and, further, a lesser amount of the inorganic salts of lime.

In rickets, which, although it is a condition affecting infancy and early childhood, affecting young bones, we have essentially a low calcium content, while the irregularity of the epiphyseal cartilage and the presence of osteoid tissue in abundance is pathognomonic.[1] However, there are likewise changes in the temporal bone which, as shown by Mayer[10] in his recent book on oto-sclerosis, consist of bony degeneration in the cartilage about the oval window, together with the same changes of ossification about the "joint" around the oval window. Mayer studied the changes in the temporal bone in a nine months old child with well advanced rickets: "The marrow, blood spaces and adjacent layers of bone stained bright red in the hemotoxylin-eosin-stained preparation, and in these red parts appeared very broad osteoid formations (bone-like tissue with deficiency of lime salts) which were sharply differentiated from the deep blue underlying bone. These changes were especially noticable in the upper layers, of the periosteal capsule. The marrow was distinctly fibrous and evident spindle-shaped cells and collagen fibrils formed a widely reticulated network in which numerous osteoblasts were seen. Another significant finding was the remains of premature cartilage in the region of the oval window, which was not ossified in the typical manner, but absorbed by the ingrowth of giant cells." Mayer quotes Pommer[11] and Schmorle who likewise showed the newly formed bony tissue remains uncalcified and covered with osteoblasts.

Ziegler also called attention to the changes in the temporal bone in rickets and to the fibrous nature of the bone marrow—an "endostitis," in which the newly formed network of bone remains uncalcified and in which the defect was lack of calcification rather than in the formation of cells.

Here, then, we have in rickets a disease of infancy and childhood, and oto-sclerosis, a condition existing between the twentieth and fiftieth year of life, changes in the temporal bone that are not dissimilar in their processes, although their end results are not quite identical. We know, furthermore, that rickets is essentially a dietetic disorder, centered on the role of vitamines and especially on the Fat Soluble A vitamine; that it can be produced experimentally by diets deficient in this vitamine, and has been prevented and cured by the administration of cod-liver oil, which contains the deficient accessory.

Can we not, then assume as a working hypothesis, that a similar deficiency acting throughout a different period of life, may not produce such a clear-cut disorder as in infantile rickets, but bony changes elsewhere?

We have, further, certain blood findings in active rickets, the most important being a lowered content of inorganic phosphorus. This finding is considered as nearly conclusive evidence of active rickets.[12] Our blood findings in this regard will be reported in detail at a later date, although in some of our cases of progressive oto-sclerosis, we have found a diminution of from 1 to 2 milligrams less of the inorganic phosphorous content per 100 c. c. In this respect it is to be noted that investigators at Johns-Hopkins University give sufficient basis to assume that the level of blood phosphate is determined in part by the amount of vitamine A available for the needs of the oragnism.[12]

Clinical application on the basis of this hypothesis has led me to give cod-liver oil to a series of patients with oto-sclerosis, particularly those in which tinnitus was a prominent symptom. In one case the tinnitus practically disappeared after three weeks and after omission of its use resumed again within a week. These cases have not been observed for a sufficient length of time to report them in detail. It is to be noted, however, that cod-liver oil has long been given in the treatment of oto-sclerosis. Siebenmann, Politzer, Hartman, Schwartze, Katz and numerous other observers, report the use of "Leberthran." It was given, however, purely on an empirical basis or for its phosphorus content,—all of these authors referring to its use in the treatment of rickets.[15] At present my experimental work, which likewise will be reported later, is concerned with the results of vitamine deficient diets on the temporal bone and internal ear of animals.

II. Hyperplastic Ethmoiditis:

Here, also, we have a clinical entity of speculative etiology, whose pathology likewise is one other than that produced by inflammatory changes with bacterial invasion. Under this nomenclature we include that type of case characterized clinically by periodical attacks of sneezing, profuse, thin, watery, irritating secretion from the nose and associated with headache, olfactory and occasionally orbital, pharyngeal or bronchial symptoms. Although no constant pathology exists, we find the middle turbinate somewhat enlarged, especially anteriorly. The bone itself may be large and easily cut off or crushed and frequently found cystic and rarefied. There are

areas of polypoid degeneration on its under or outer surface and later polypi may become very manifest within the nose. The ethmoid cells are usually distended with polypi and many partitions are thus destroyed by pressure.

Histologically, the osseous changes are those of absorption, the bone thickened because of a rarefying process. These changes may or may not be secondary to those of the overlying mucous membrane. The latter shows metaplasia of the ciliated epithelium; the mucous glands may show hypertrophy primarily and not infrequently cystic dilatation. The sub-epithelial structure show fibrous changes and the exudation of serum into the epithelium and stroma is prominent.[13]

It is seen, therefore, that the changes are apparently due to some disturbance in nutrition of the ethmoid capsule, as suggested by Skillern.[14] What relation this condition may have to a deficiency disease is, of course, hypothetical. There is seen, however, pathological changes that are not unlike those found in other well-defined deficiency diseases as shown by McCorrison.[3] One group of typical cases was, therefore, given a dietary rich in water soluble B and C vitamines, while in the second group there was simply added to their dietary, cod-liver oil which is abundant in the fat soluble A vitamine. The first group showed no improvement after a period of observation, while the second group after an average period of three weeks began to show definite amelioration of symptoms. It might also be mentioned here that a third group of these cases was treated solely by the ultra-violet rays with correspondingly good results. This plan of treatment seems to be in accordance with the view of Ludwig Meyer[16] who believes that in rickets, for instance, the dietetic and the physical methods of treatment reach the same end although from different angles. Observation on these cases has not been of sufficient length to warrant any further detailed report which will be submitted, however, at a later date together with our blood findings and experimental evidence in animals.

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EPIGLOTTIDECTOMY FOR THE RELIEF OF CONGENITAL LARYNGEAL STRIDOR, WITH REPORT OF A CASE.*

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Congenital laryngeal stridor is a rare condition, characterized by its onset shortly after birth and by a peculiar inspiratory obstruction associated with a crowing sound. The stridor usually disappears during the second year of life. Dyspnea is usually absent, but there may be considerable inspiratory retraction of the lower thorax and at the sternal notch. In severe cases, asphyxial attacks may occur and may at times be so severe as to cause death.

Thus Reardon,[1] in reviewing 101 cases found that "in seven cases death seemed directly due to the stenosis." While Blackader and Muckleston[2] have found that one-sixth of the cases reported have died from affections of the respiratory system.

According to Thomson and Turner,[3] the infantile epiglottis is characterized by its soft non-resistant texture and by more or less approximation of its lateral edges and of the ary-epiglottic folds. This gives the epiglottis a peculiar trough or gutter-like shape. As the child grows the epiglottis gradually becomes more rigid and elevated and tends to unfold, so that by the ninth year it begins to assume the form of the adult epiglottis.

In cases of congenital stridor most observers have found an exaggeration of the infantile type of epiglottis. In such cases, upon examination, the epiglottis can be seen to tilt backward, during inspiration, while its lateral borders and the aryfolds frequently come into contact. The upper aperture of the larynx may thus be reduced to slit-like orifice with a rhomboid opening just anterior to the arytenoids. The stridor is probably due to the vibration of the aryfolds and the borders of the epiglottis.

Post-mortem examination of the larynx from stridor cases shows that this deformity persists even after death, and it seems most likely that the malformed epiglottis is the cause of the symptoms. Jackson[4] states that occasionally the epiglottis of normal infants has a tendency to roll inward during inspiration but considers the stridor cases a pathological exaggeration of this phenomenon.

Lees [5] was among the first to demonstrate such a larynx. The illustration of this specimen shows the epiglottis markedly in-

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curvated with its lateral halves in contact, and with the laryngeal vestibule reduced to a mere slit. Variot[6] demonstrated a similar specimen in 1898.

Judging from the illustration of Refslund's[7] case the pathological anatomy was almost identical with Lees' case. The autopsy in Koplik's[8] case, in a one-year-old child, which died of pneumonia showed a pronounced infolding of the epiglottis with its edges in contact. The lumen of the larynx seemed narrower than normal. An enlarged thymus was also found in this case. Variot[9] has recently found some abnormalities in the muscles of the larynx of a child which had presented symptoms of stridor during life. The right crico-arytenoideus posticus muscle was entirely wanting, and there was slight atrophy of the vocal cord.

Report of Case: About five months ago a pronounced case of laryngeal stridor came under my care. The case is of special interest because the infant was born in the hospital, where it was carefully observed by Dr. Blackfan and Dr. Higgins, of the Pediatric Service. Further interest is added by the fact that the infant had an enlarged thymus to which the symptoms were at first attributed. The thymus was treated by Dr. Little with the X-ray and as a result diminished markedly in size, but the symptoms persisted.

The following abstract of the history prior to the time I saw the case is quoted from Dr. Blackfan's [10] records: "A colored female infant weighed six pounds at birth. (Born in the Cincinnati General Hospital, March 29, 1921.) Immediately following delivery it was observed that there was difficulty in breathing. There was marked inspiratory stridor and cyanosis. The dyspnea was constant and from time to time it became paroxysmal; being increased by crying and nursing. The paroxysms would begin with a series of inspiratory efforts followed by a loud crowing inspiratory sound and then followed by a short expiratory effort. During this there was marked retraction of the supra and infraclavicular spaces and the intercostal spaces. There was an anxious expression of the infant, the eyeball became prominent and there was extreme cyanosis. On several occasions the attacks were so severe as to require artificial respiration.

The physical examination was negative except for an area of increased dullness in the region of the thymus. The dullness extended one-and-a-quarter inches to the left and seven-eighths of an inch to the right of the mid-sternal line.

It was continuous below with the cardiac dullness. This area of dullness disappeared when the infant's head was held in extreme

dorsal flexion. It was also noticed that the symptoms were markedly relieved when this position was assumed. It was repeatedly observed that the symptoms could be induced and relieved by the position of the infant's head. When first seen it was believed from the increased area of dullness that the symptoms were due to an enlarged thymus. The possibility of congenital malformation of the larynx was however considered. In as much as the X-ray picture showed a shadow in the upper mediastinum indicative of an enlarged thymus a series of X-ray treatments were begun. Although no marked improvement in the symptoms followed this treatment the shadow in the X-ray entirely disappeared. In as much as the infant was being breast fed and the paroxysms were less frequent, the patient was discharged after one month," with instructions that it was to be brought to the dispensary at regular intervals.

Ten days later, on May 4, 1921, while in the dispensary the child suddenly became cyanotic, ceased breathing and required artificial respiration stimulation to restore it (Dr. Higgins). On the next day I noted the following: Voice normal, as manifested by crying. Respiration interrupted at times by laryngeal stridor with supra and infraclavicular retractions and girdle-like retraction of the lower ribs. On direct examination of the larynx, the free borders of the epiglottis are drawn together at each inspiration and come into contact; at the same time, the epiglottis is drawn backward into the larynx. The epiglottis appears to be flabby in consistence. The epiglottis closes like a book during inspiration. Arytenoids and vocal cords appear normal in structure and motion.

Epiglottidectomy: On May 6, 1921, without anesthesia, I suspended the patient, using a very short spatula made overnight especially for this case by Dr. Henry Freiberg. The epiglottis was exposed by the suspension laryngoscope. The tip of the epiglottis was seized with a small aligator forceps and a nasal snare was passed over the epiglottis, severing the free portion. There was practically no bleeding. It was rather soft and flabby but showed no gross anomalies. (Histological examination of the specimen by Dr. Austin showed nothing abnormal in structure.) The child was able to swallow in a normal manner on the same day and thereafter. The stridor, however, did not disappear immediately, but recurred at times for about twelve days after the operation, after which it was no longer observed. No asphyxial attacks occurred after the operation.

Five months after the operation the child breathes in a perfectly normal manner, when quiet, but shows a slight retraction

of the lower ribs when it cries loudly. The mother states that it has never had any difficulty in swallowing and she has not noticed any stridor except when the child has a cold. A very small epiglottic stump can be made out on direct laryngoscopy.

Comment: Since most observers agree that congenital stridor is due to a valve-like infolding of the epiglottis it seems to be a rational procedure to remove the epiglottis in all cases subject to asphyxial attacks. As is well known the epiglottis can readily be dispensed with in older patients and its removal apparently has no influence on deglutition even in infants. The persistence of some stridor for several days after the operation described above is difficult to explain, but may have been due to inflamatory reaction; or the flopping of what was left of the ary-epiglottic folds after the epiglottidectomy; or to some weakness in the laryngeal skeleton. At any rate the stridor disappeared much sooner than could have been expected in the natural course of the disease.

As an alternative to epiglottidectomy tracheotomy or intubation must be considered. Epiglottidectomy is a much simpler operation than tracheotomy with less risk to the patient, while intubation even if efficacious would require the wearing of a tube for many months.

So far as I can escertain, epiglottidectomy has not hitherto been performed to relieve congenital laryngeal stridor. A review of the literature for the past twenty-five years apparently contains no record of such an operation for this condition. (American Institute of Medicine). This is more to be wondered at since Variot,[9] in 1898, suggested that cutting away the ary-epiglottic folds might relieve this condition; and Reardon, op. cit. in 1907, suggested that the removal of the incurvated portion of the epiglottis probably would give relief if the removal of the epiglottic folds did not suffice.

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THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

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When Shall the Membrana Tympani be Incised and When is Re-incision Indicated? Dr. Walter Lester Carr.

Dr. Walter Lester Carr presented this paper, in which he urged that a routine examination of ears and throat be made in all infectious diseases, and in every condition where there was a question as to the cause of irregular high temperature with or without local pain in the ear. A child might have pain without a rise of temperature, or at most a degree above normal, or there might be restlessness or irratibility without temperature and pain. Examination must be made with a good reflected light after the external canal had been wiped clean. The necessity for this examination is shown by the fact that every physician is liable to be chagrined by the report that "the ear burst in the night." He had had such an accident in children with acute piliomyelitis and in bronchitis when he had not anticipated it. If the tympanum was found to be red and bulging, the decision to open the drum should be made at once, especially if the child had a rise in temperature and pain. When there was congestion and bulging, as was seen in scarlet fever, the advisability of myringotomy might be determined, in part, by the state of the pharyngeal tissue. If the structures of the tonsils and pharynx were reddened and swollen so that there was blocking of the outlet of the Eustachian tubes, an incision of the drum might be advisable. It might be possible to abort such a condition by the use of adrenalin solution, 1 part to 4 or 5 of normal saline solution instilled into the nares so that it came in contact with the swollen Eustachian tubes. An incision of the drum was not, however, the whole treatment. In addition to irrigations of the external auditory canal, local applications to the pharynx and tonsils of argyrol or a similar preparation were essential to get the best results of drainage from the cavity of the middle ear. Where there was an acute infection of the middle ear, as in pneumonia, tonsillitis, influenza, etc., the pediatrician was almost always willing to incise the drum or have it done by an otologist as he realized that to allow the infectious process to damage the structure of the middle ear would cause changes in the drum membrane and ossicular joints and might allow the disease to extend to the mastoid process. Although a patient could not be protected against a mastoid disease which might be occasioned by a systemic infection, it was possible to give some drainage through the external meatus. In children who had an earache, associated with mild catarrh, naso-pharyngitis, hay-fever and gastro-intestinal disease disturbances, decision to open the drum should be determined by the symptoms of the individual child. One who was nervous and high strung and who had pain and slight elevation of temperature might be better for having the ear drum incised, even when there was not much bulging or congestion. Many nervous children got comfort from the application of local heat, of argyrol to the nasopharynx, and instillations of warm boric acid solutions into the ear. Medicinal agents, as calomel, were of service, and small doses of codeine, antipyrin, sodium bromide and belladonna gave comfort. The instillation of warm oil was not so helpful and might be detrimental. The writer had found some benefit when novocaine and adrenalin solutions had been dropped on a congested tympanum, but he had not depended upon these agents alone, and was unable to state how useful they were. In his experience patients who had relief only when chloroform vapor was blown into the ear required incision. At the New York Eye and Ear Infirmary the

organisms found in smears after incision of the drum were streptococci, pneumocci of the different groups, Friedlander's bacillus, staphylococci, and B. catarrhalis. When cultures are made, care must be taken in method and media, or staphylococci might overgrow other organisms and give rise to a faulty interpretation. Reincision might be required if a primary incision had not been free enough for drainage and when infection and swelling of the Eustachian canal blocked the way to the pharynx. Reincision was indicated particularly when there was sagging of the wall of the external auditory canal, even if no other symptoms of mastoid disease were present. Reincision must be made if the tympanum shows the same condition that necessitated primary operation, namely, pain, bulging, and elevation of temperature. Reincision might be indicated in children of poor vitality where drainage from the middle ear was imperfect, even after removal of hypertrophied tonsils and adenoids. In all children in whom an incision had to be made in the drum membrane there was no excuse for omitting the removal of tonsils and adenoids, even when these structures did not show a great degree of infection and obstruction. Children of low vitality needed also hygienic and tonic treatment. There were cases in which mastoid symptoms were present in which the otologist and pediatrist would have to decide whether the incision should be made or the mastoid cells excenterated.

Limitations of the Re-incision of the Tympanic Membrane. Dr. Samuel J. Kopetsky and Dr. Alfred A. Schwartz.

Dr. Samuel J. Kopetsky and Dr. Alfred A. Schwartz presented this communication from the Oto-Laryngological Department of Beth Israel Hospital. They stated that employed early and properly performed the value of incision of the membrana tympani as a surgical procedure admitted of no discussion. Due to factors other than the establishment of drainage, a certain percentage of cases developed into the type of mastoiditis calling for operative interference in spite of early drum incision. In an effort to prevent this development, a great many physicians—and in this class must be included a number of otologists—incised the drum membrane repeatedly. They argued that if it were true that simple incision of the membrana tympani gave complete relief in certain cases of otitis media, then surely in cases in which the disease showed a tendency to spread further than the confines of the middle ear and antrum a second incision might increase the drainage obtained, and so they continued on until perhaps shredding of the drum was accomplished in the vain and illogical effort to drain diseased regions which were out of reach of surgery applied to the middle ear. The writers contended that paracentesis or repeated paracentesis could not prevent or cure operative mastoiditis. Only in young infants could reincision of the drum-head be logically employed, and this because of the rudimentary and undeveloped mastoid process. Bearing in mind this one exception, their contention found further support in a study of the pathology of mastoiditis, the mode of infection and the clinical course of acute purulent otitis media. Did one but visualize the stages and evolution of the pathological lesion in every case as it was presented to him, the chances of misjudging the necessity of repeating incision of the drum-head would be lessened. Too often the clinician does not do this. It was of no avail to repeatedly incise a drum-head to evacuate pus and to attempt to prevent an operation on the mastoid process in the type of case whose clinical history, bacterial flora and general aspect were such that one was reasonably certain that it fell within the group-type termed "hemorrhagic mastoiditis." In this type the infection was not walled off as in the coalescent type, but the dilated blood vessels rapidly carried the infecting organisms throughout the mastoid and, unless the process was promptly and completely checked, even further, into the sinus, meninges and brain. This form had been rightly called the dangerous form of mastoiditis, and its prompt recognition was imperative for the preservation of the patient's life. Yet, strange as it might seem, it was this type

that was most frequently overlooked by the attending physician. The patient complained of pain in the ear, and there was a high temperature, 103 or 104°. Paracentesis was promptly and properly performed, a free incision being made into the membrana tympani. Much to the surprise of the attendants on the case, there was only a slight seropurulent discharge from the middle ear, the temperature had again gone up and the patient was distinctly ill. There might be slight mastoid tenderness, not sufficient to occasion alarm. Another paracentesis was performed, then perhaps a day later, another. In the meantime the mastoid infection had continued unchecked, and complications with symptoms referable to the sinus and meninges put in appearance. This picture was not overdrawn, for it had been observed time and time again during the past two years, especially during the recent influenza epidemics. In these cases valuable time had been lost, repeated paracentesis had not prevented the operative mastoiditis and the patient's life had been greatly endangered. A slight discharge from an incision of the membrana tympani might mean not an insufficient opening, but a virulent infection. In this type of mastoiditis there should be borne in mind another factor, namely, the simultaneous involvement of both middle ear and mastoid where both demand surgical interference from the onset. In the so-called coalescent type of case, where drainage is profuse and inspection reveals that the incision of the drum-head is adequate, and the pus comes away in pulsations, too profuse to be considered as coming from the tympanic cavity, nothing was accomplished by reincising the drum, and other problems than drainage were demanding solution. The fact to be stressed was that the physician should familiarize himself to a greater extent with the types of acute purulent otitis media and of mastoiditis. Let this knowledge become more general, and agreement upon the surgical procedures to be employed would soon ensue. When, after the delay occasioned by the attempts to cure the mastoid disease by reincisions, the patient finally came to the opertation, the disease might be far advanced, with marked destruction of the mastoid structure, and the drum might be shredded or practically destroyed. Hearing was markedly impaired in many cases treated by reincision. It had been claimed that the drum membrane was essential to hearing. Educational tests proved the fallacy of this contention. In a cursory examination of their office records the writers had found as many as eight cases that eventually came to operation after having been previously subjected to three, four or five paracenteses each before coming under their care; and in some of these all the paracenteses were made during the first seventy-two hours. Not only was mastoiditis not prevented in these cases, but in three septic throbosis developed, and one died of a suppurative meningitis. A large number of their cases of chronic suppurative otitis media traced their origin to an acute attack where repeated paracenteses were performed. Many were now in such a condition that they could not be cleared without the radical operation. There were others where the drum membrane had healed and the middle ear was dry. The multiple scars were frequently adherent to the middle ear structures and there was loss of mobility of the ossicles, resulting in marked impairment of hearing and tinnitus. It would have been much better in these cases to have drained the antrum from behind or eradicated the disease in the mastoid process and to have prevented the chronic diseases from gaining a foothold. For a simple mastoidectomy should leave the hearing unimpaired and markedly diminish the risk of further complications.

DISCUSSION.

Dr. John R. Page said that Dr. Carr had stated the situation very well indeed from the standpoint of the otologist and that he had no disagreement with the views expressed. He had been led to believe that most children's men were decidedly less radical, if those statements were considered radical. The otologist and the pediatrician disagreed on this

question for the same reason that they did not always agree as to when it was best to open a mastoid, and this was quite natural. The ear man, from his association with complicated cases, has the danger of delay impressed upon him so forcibly that he would rather chance erring on the safe side than open too late, feeling, as he does, that harm practically never results from an early paracentesis. The children's man doubts this, and advances as an argument against it the danger of infection from outside, having in mind when he does, not a paracentesis, but a free incision for drainage—a U-shaped incision, etc.—so much talked about, but so seldom performed, while the ear man has not this in mind at all. He looks at the ear drum, and if he sees evidence of marked involvement with infection, he makes a free incision from below upward in the posterior part of the membrane and evacuates the contents, but if he finds the membrane retracted and reddened, with the patient in pain, he does a paracentesis (nothing more) with a small knife, which is sufficient to relieve the pain and let any extravasated serum escape that may be present. The early cases of otitis media have a negative pressure in the tympanum, and in some cases the pain is as acute at this stage as it is in others from fluid under pressure. Just as a dry cup produces a hyperemia of the skin and—if its use is prolonged—hemorrhage and exudation, so will negative pressure in the tympanum cause swelling and congestion of the mucous lining and extravasation into the cavity. A minute opening into such a drum relieves the situation immediately and almost invariably. The chance of carrying infection through a thin membrane into such an air chamber with a sterile knife is very slight and not to be compared with the danger of lumbar puncture, so often considered harmless. Of course, if a U-shaped flap is going to be made and a fluid syringed through into the throat, as some think advisable, any careful man would rather take the chances with nature than to subject the child to such heroic treatment.

In any case in which the membrane can be seen through a speculum, the canal can be cleansed and filled with alcohol and the drum entered with the point of a small sterile knife with no more danger of outside infection than in a lumber puncture, and it is surprising to learn that beyond the knife and the speculum these precautions are not always regarded as necessary by some who perform myringotomies.

In cases where the canal is so swollen that the membrane can hardly be seen and the knife has to be passed in contact with the posterior wall as a guide and the drum opened blindly, then, of course, there is a greater chance for infection, but these are cases in which the process is usually well established and there is little doubt of the necessity for incision. Still, even where the swelling is marked, packing the canal with cotton moistened in alcohol and allowing it to remain in place for five minutes will squeeze out the edema and give a good view more often than is realized by many.

A paracentesis or myringotomy cannot be performed satisfactorily with the infant in the upright position, for in that position one cannot properly see what he is doing nor properly cleanse the canal, and if the child, when tightly swathed, cannot be held still on the table, it can be given a little whiff of chloroform unless there is some very positive reason to the contrary.

Dr. Page said that the efficacy of the simple puncture done early had been demonstrated more than once in his own family when he was on hand at the beginning of the trouble; and in one instance the danger from delay was also demonstrated when his child, being away from home, a double otitis developed, as it had done before on several occasions: this time it was allowed to become established because a very good doctor, for 48 hours, thought the membrane was not bulging sufficiently to warrant opening. A double mastoiditis developed which barely escaped operation, and marked impairment of hearing and tinnitus persisted for months.

Dr. Page did not wish it to be understood that he opened every drum membrane that he met. On the contrary, he frequently advises waiting, but keeps in close touch with such patients and will not allow the ear to be irrigated with hot water so as to render it impossible to tell how much the increased redness is due to the hot water at 110° F. It is fortunate for patients that the directions for hot irrigations are usually so poorly carried out that the water does not reach the drum, for it is liable to make them dizzy and seasick from the caloric action on the labyrinth. The canals, however, are usually swollen by it and it creates doubt about the membrane itself. A hot water bag masks matters less and while it does not divert the patient to the same extent, it probably affords as much relief.

Dr. Page said further that he wished he were able to get as much relief through intranasal drops of adrenalin, argyrol, etc., that some seem to secure. Despite the opposition, he felt that he obtained more certain ventilation of the drum and more prompt relief from pain by the small, precise, careful and—as Dr. Gruening used to say, "in my hands"—harmless perforation of the drum.

In regard to re-incision: It may be necessary a day or two after the first paracentesis if in an ear where the infection is well established, the drainage is insufficient and there is some doubt as to whether a proper incision was made, to meet the condition in the first place. It can do no harm and it frequently does good, but repeated incisions made simply because the drum bulges are useless in a well infected ear. A membrane can be cut to ribbons and it will still bulge, not from retention, but from swollen mucosa in the region of the additus and from granulations in the tympanum.

Without invading the topic to be discussed later, Dr. Page said that a word about persistently discharging ears was justified just here, and he appealed to the children's men in hospital practice to note all running ears in children that have been cured of the various diseases for which they were admitted, and urged that the House Physician should stamp on the cards of these patients that they have a purulent otitis media and should be referred by the office to a proper place for observation and treatment. If this were done there would be fewer cases turning up with the membranes sloughed out by prolonged discharge.

Dr. Wendell C. Phillips, referring to Dr. Page's paper on the subject of paracentesis, said that there were a few points that might be brought out more clearly. He could not himself follow Dr. Page in all of his contentions. By a long experience he had been led to believe that there are two distinct types of earache occurring, not only in children, but also in adults, one, a lancinating type, and the other a dull pain in the ear. The one is catarrhal otitis media acuta, and the other is the purulent acute type. The catarrhal type is not an infection, but is a mechanical condition caused by obstruction of the Eustachian tube, and in that case the pain is due to negative pressure and the ear drum, while red, is retracted. "No matter how red and long-continued that case, I would keep the knife away and not make any incision. The other type is the purulent type with severe inflammation and a bulging drum. In the purulent type an early paracentesis is desirable. I cannot help feeling that Dr. Page sometimes opens a drum that is not purulent. A teacher has to be somewhat dogmatic, and I have always taught that the catarrhal type is always catarrhal and never purulent; the purulent type is always purulent and remains purulent until it is relieved. As a rule, in the purulent cases when you have done a free paracentesis you will not obtain much benefit by a repetition of the operation." Once in a while it may be done, mainly to appease the family, but repeated opening of the ear drum is simply a postponement of the time when the operation must be performed.

Dr. Miner C. Hill said that it always seemed to him that when in doubt paracentesis was a safe procedure and often relieved symptoms, even when there was no pus in the middle ear. Pain has been mentioned

as an early symptom. In most children pain is often not mentioned and the symptoms suggesting otitis media are fever, irritability and cough. It would be interesting to hear the experiences from some of the pediatrists on this point. In the last influenzal epidemic we saw numerous cases of acute otitis media, without symptoms, which cleared up without any incision at all. This would indicate that a red drum does not always indicate incision.

Dr. L. E. La Fetra said that it had always seemed to him that the otologists were rather pessimistic in the presence of a bulging drum membrane. All agree that if a child has a high temperature and the bulging of the drum membrane persists beyond twenty-four hours that the membrane might well be incised, but so many of these cases clear up even when the bulging is general, that he could not believe that the presence of a bulging drum membrane was an absolute indication for a paracentesis. In an experience with an enormous number of cases of acute otitis media, many of which were not incised at all, he had seen so many clear up without a paracentesis that he could not take quite the radical view of many of his otological friends. If, after 24 hours, there is still bulging of the drum and high temperature, one should not wait longer, for the dangers of mastoid are so much greater than the danger of a mere incision of the drum that no one should hesitate.

With regard to a second incision of the drum membrane, he was inclined to agree with the otologists that if a good free opening is made the first time and fails to get relief of the symptoms, and the discharge persists, it is evident that something more is going on behind the membrane and one should look for trouble in the mastoid. If, however, the opening in the drum heals rapidly, re-incision should be done, and done repeatedly. A point to be emphasized in regard to children is that the presence of a vacillating septic type of temperature with a high leucocyte and a high polynuclear count does not necessarily mean a mastoid, nor a sinus thrombosis; one must be guided by the otological signs rather than by the temperature and the single blood count.

Dr. Schwartz said, that due to factors other than the establishment of drainage, a certain percentage of cases of acute otitis media develop into the type of mastoiditis calling for operative interference in spite of early drum incision. Re-incision of the membrana tympani cannot prevent this complication, except, perhaps, in infants.

This contention finds support in the study of the pathology of mastoiditis, the mode of infection and the clinical course of acute purulent otitis media. In the "hemorrhagic type" of mastoiditis there is a virulent infection, carried by the blood stream, and the infection is not walled off. Valuable time may be lost by re-incising the membrana tympani. In the coalescent type there is a true abscess formation in the mastoid process and re-incision of the drum cannot give more room for drainage than the single free incision.

The after effects of re-incision—unhealed perforations, marked scarring of the drum and the symptoms due to adhesions in the middle ear—make their appearance later in life.

Not only has not re-incision of the membrana tympani prevented operative mastoiditis, but in our recent records there are a number of cases where delay has resulted in septic sinus thrombosis, and one case died of a purulent meningitis.

Dr. Schwartz claimed, therefore, that re-incision of the membrana tympani cannot prevent or cure an operative mastoiditis; that it shreds the drum and frequently leaves the beginning of a chronic purulent or chronic "catarrhal" otitis media, and that the delay occasioned by the frequent incisions may give time for the infection to spread to the sinus, meninges or brain.

Dr. C. H. Smith agreed with the otologists that it was useless to shred an ear drum two or three times in the first stage of the disease, but in a certain number of cases in young children the first incision will heal before the otitis has entirely resolved. At the end of ten days

these may require re-incision, and get well after it without mastoiditis. This is a common type in children's wards and also in private practice.

Dr. Koplik, referring to paracentesis, said that as a man who examines the ears of almost every patient who comes to the office and who consequently sees various types of otitis, his view was naturally that of one who considers not only the ear, but the baby's comfort and welfare. It is his practice to examine every ear, no matter what is the matter with the baby. There are cases of otitis without temperature, absolutely no temperature above 100°, and the babies are very sick, and if you examine the ear you find pus; there may be very little bulging, but you find pus; there may be a temperature of 98.5° and a serous exudate in the ear. The patients have been sick and weak with very few symptoms, and the drum may not be bulging, but flat, and when the drum is opened you find pus. In other words, the baby's comfort is very important, and it should not be allowed to have to wait twenty-four hours for the drum to go down and pain subside. If a drum is bulging, he does not hesitate to advise incision. Although in some of these cases if the ear is let alone it may resolve, yet in many of such cases there is trouble later. If the otologists will recall the cases they have seen, they will remember many where the drum did not bulge, but has been rather flat, yet there has been pus in the ear.

In regard to re-incision, Dr. Koplik said that he and a colleague, an otologist, had had many cases where a mastoid operation had been advised, but which had been cured by simple re-incision, with very good drums, and the children remained perfectly well. The ears may have had to be re-incised once or twice in order to obtain good drainage where there was a damming up, but the babies got well without the mastoid operation which had been advised. Of course, there are cases where it is not well to take chances, and one must have a large experience and be able to judge each case as an entity.

Dr. Quinlan expressed his gratification at the collaboration of the pediatrists and the otologists, for the topic was of great importance to both, and said that he had listened to the papers and discussion with much edification and pleasure. He has not now so much of the enthusiasm of youth as he has the wisdom of age. He had listened to Dr. Page with much satisfaction and a little suspicion, and noted that he said his children were particularly affected at night with this earache. In years gone by it was learned that night earache is probably due to the recumbent position owing to an occlusion of the Eustachian tube from the pharynx to the middle ear, the tubal muscles become edematous and obstructs the passage of the air—simply a hyperaemia of Schrapnell's membrane. That would not seem to require incision, for such a measure means much, and unless a man has great accuracy of sight and a wonderful touch he is apt to injure the incus and put the patient's hearing out of business for the rest of his life. If a prophylactic measure was resorted to—say the depletion of the Eustachian tube by the application of a 2 per cent solution of silver—it would empty the engorged tubal condition and re-establish the circulation of the air in the middle ear. This happens very frequently with cases that are hyperemic at night; it occurs as the result of some lymphoid tissue around the pharynx or Eustachian tube which interferes, causing a stasis. These cases can be treated without a knife or any heroic measure.

Dr. Quinlan agreed with Dr. Phillips that when one sees a drum pushing down and a sagging condition of the canal wall reveals the presence of a purulent discharge, then and not until then, is it wise to incise it.

Dr. Sidney Valentine Haas said it took some courage to speak against early paracentesis. Dr. Le Fetra and Dr. Quinlan had advocated conservatism, and he would like to tell something of his personal experience beginning fifteen years ago. It was taught at that time that unless the drum was incised early a mastoid operation would probably become necessary. In one family there were three cases of otitis media which

were opened within the first twenty-four hours, and two of these had to have a mastoidectomy. That made him believe that if one waited for a short period it might be no worse. From that time he began to wait, increasing the intervals, and at the end of ten years he had no hesitation in waiting days. He was sure *early* paracentesis was not only not indicated, but actually harmful. It was better to wait three, four, or five days than to make the incision within the first twenty-four hours. For the pain there was nothing that worked better than opium. Certainly the taking of an anesthetic was as bad as the short period of pain, which could be relieved by paregoric. Incidentally the primary pain when relieved only rarely recurred. He was speaking not only of congested ear drums, but also of tender mastoids in patients with a high temperature. Dr. Page would perhaps recall a case that they had seen together. Dr. Page was called in on the fourth or fifth day to do a myringotomy and had objected greatly to the delay. The infection in this case was a *streptococcus mucosus*, and although the mastoids were involved, they cleared up quickly. In the last four or five years he had not had a case of operative mastoiditis by following this policy of waiting. This but it did show that complications were not incurred in waiting. It was did not mean that he would not have any cases of operative mastoiditis, quite remarkable to see how these bulging and inflamed drums would subside without incision under hexamethylenamine and argyrol. Finally one could judge by the appearance of the nasopharynx as to the progress of the process in the ear. The temperature dropped to normal long before the bulging subsided. The improvement in hearing was also a guide as to the conditions existing in the middle ear. There had been no case of later impairment of function.

Dr. Seymour Oppenheimer expressed the opinion that they ought to be able to strike a sane attitude on this subject. In his judgment myringotomy was performed too frequently. He observed so often the children admitted to his hospital service with some pulmonary infection or some intestinal disturbance and almost uniformly the ear drums of these children had been opened by the house staff or visiting staff.

Most of these cases had reddened drums, but that did not indicate the necessity for myringotomy. The only indication is the evidence of an exudate in the middle ear space; indication may occur on the first, second or even the tenth day. The temperature does not enter into it, but it is the appearance of the drum membrane which is the guide.

Exudates ofttimes form very rapidly and at the time of examination a myringofomy does not seem necessary, and then some hours later one is mortified to find that a perforation has occurred; that is something that cannot be helped. As to the question of re-incision and its necessity, much depends upon the type of treatment employed after the original myringotomy, provided, of course, the myringotomy has been properly performed. Unfortunately there is an impression that myringotomy consists in nothing more than a stab. It should be an accurate surgical procedure and properly placed. The after treatment is most important to keep the perforation patent and the middle ear freed of secretion, and for this purpose the employment of negative pressure by a simple suction apparatus is most satisfactory. If that type of treatment is employed the necessity for re-incision of the drum would be much lessened. Doctor Oppenheimer said he was sorry he could not agree with Dr. Haas. They had been threshing that question out together for many years. While he believed that drum membranes were sometimes opened too early, he was not in accord with Dr. Haas's views. The question of the virulence of the particular infection in the individual case is all important. The question of proper attention to the canal of the ear is of the greatest importance and it is distressing to see men pay such little attention to asepsis and the surgical preparation of the ear canal and the ear proper prior to the incision. This brought up the question as to whether infection could be carried from without. Dr. Oppenheimer firmly expressed the opinion that it can unless proper aseptic precautions are employed.

DR. PAGE, in closing the discussion, remarked that there seemed to be quite as much difference of opinion among the otologists and among the pediatricians themselves as there was between the otologists and the pediatricians. There was equal support on both sides.

Factors Determining That a Simple Mastoidectomy is Indicated in the Non-Acute Stage of Middle Ear and Mastoid Infections. Dr. Henry Koplik.

A Further Report on the Treatment of Persistent Otorrhea in Infants and Young Children by the Establishment of Postauricular Drainage. Dr. Wendell C. Phillips.

About eight years ago I had occasion to outline the results of a series of studies regarding the more or less chronic purulent middle ear suppurations occurring during infancy and early childhood. Those studies were based upon a large clinical experience, both in private practice and in my various hospital services. At that time I defined two distinct types of this particular infection as follows: (A) The persistent type, and (B) the recurring type. The persistent type applies to those cases in which an acute purulent inflammatory process invades the middle ear spaces, and continues without abatement for an indeterminate period after the usual five to thirty days, which ordinarily may be considered as its normal course. This type of purulent otitis media should be clearly differentiated from the more common and recurrent type in which recovery usually takes place from each attack, only to be followed by recurrence at varying intervals, or whenever the child becomes the victim of an acute nasal infection. In the recurrent type the children are almost invariably victims of diseased tonsils and adenoids, and their attacks usually cease upon the removal of these offending tissues. The recurrent type, therefore, may be eliminated from this discussion, inasmuch as it hardly may be classified as non-acute in character unless, as a result of neglect to remove the diseased tonsils and adenoids, the otorrhea may finally become persistent and chronic in character. The persistent type of cases furnish a history somewhat as follows: During the course of an infection involving the upper air passages, or as a complication of certain chronic diseases, an acute ear suppuration appears. Weakened by the primary general acute infection, the child's resistance becomes lowered and in many instances it is probable that some underlying dyscrasia, known or unknown, plays its part. Earache follows, which may be relieved by a paracentesis, and from the onset the discharge is profuse, persistent, and of a character which plainly bespeaks a severe type of infection.

Mastoid tenderness is present and positive during the first few days, but may gradually disappear, unless the case is destined to become an acute mastoiditis requiring immediate operation. A large percentage of cases, however, show a moderate diminution in the quantity of the discharge from the third to the seventh day, but it never entirely ceases. The temperature may gradually become normal, or nearly so; pain is not present thereafter as a prominent symptom, and there may be no marked drooping of the postero-superior canal wall. The usual local measures of treatment, consisting of paracentesis, cleansing, cathartics and rest in bed may have been carried out, but often clinic patients have received no treatment at all. In short, the case becomes one of chronic purulent otitis media. Its significance being otorrhea, which continues without abatement. To the observer it becomes evident that the small tympanic cavity proper could not secrete the amount of pus which flows from the ear, and that the mastoid antrum at least must be involved in the inflammatory process. Furthermore, it is evident that when such a discharge continues for a period beyond the end of the fourth week, the element chronicity has become established. It is also true that proportionately a large number of these persistent otorrheas occur among the poor and ill-nourished applying for treatment in the hospitals or dispensaries.

Of the underlying dyscrasias which serve to prevent nature's efforts at repairment, tuberculosis and syphilis are the most stubborn to deal with. Examinations of the discharge and scrapings from a considerable number of this type of patients were examined for tuberculosis with positive results, even in cases where no general evidence of tuberculosis existed. In this particular type of otorrhea even the removal of the tonsils and adenoids does not serve to terminate the aural discharge; neither does repeated paracentesis serve any purpose. The disease remains chronic and unless relieved by surgical measures it progresses for long periods with serious impairment of hearing and often with fatal complications. It may be true that after long periods, even years, some of these cases do discharge spontaneously, but not until after extensive destruction has taken place with permanent impairment of the hearing.

Regarding the treatment: All reasonable local measures should be faithfully carried out for a reasonable period, which may be from a month to six weeks, this to include free drainage by paracentesis, a clean regime of local treatment and attention to hygiene and the general health. All these measures having failed, it is my opinion that the quickest, safest and most rational measure to pursue is to establish post-auricular, and hence, through and through drainage by means of the simple mastoid operation. This was the conviction expressed eight years ago, and this opinion was based upon about 20 or 25 operations. The lapse of years and the further study of cases, together with about 100 operations performed by myself and assistants, has in no way changed this conviction. In well defined cases of chronic otorrhea of the type above defined I have never failed to find disease of the mastoid process and have rarely failed to note the free escape of pus through the opening in the cortex at the time of operation. It must be remembered that these cases fail to exhibit almost all of the classical symptoms of mastoid involvement, except the discharge; yet, upon operation all furnish evidences of extensive disease of the mastoid cells. Briefly stated, the reasons for advocating the simple mastoid operation in these cases are as follows:

1. It quickly terminates an otherwise persistent otorrhea.
2. It insures against an extension of local bone necrosis.
3. It relieves the patients of the dangers of possible serious and fatal complications, such as meningitis, brain abscess and lateral sinus thrombosis.
4. Finally, the most important reason of all is the retention of the hearing function.

It is scarcely necessary to add that this operation is not a dangerous one; neither is its slight seriousness to be compared with the dangers and discomforts attending a chronic aural discharge, and particularly the threatened deafness. It should be added that in the more chronic cases the simple mastoid operation has occasionally proven insufficient and a subsequent radical operation has become necessary.

DISCUSSION.

DR. MEIERHOF continued the discussion as follows: When has an infection of the middle ear, accompanied by a flow of pus, become acute in an infant or a young child? Is this to be determined by the absence of reactionary symptoms—principally fever and evidences of pain, either spontaneous or induced by pressure over the mastoid—or by traction upon the auricle, sometimes accompanied by restlessness during the sleeping period? If such be the case one would have to wait a very long time in many cases before the non-acute stage has arrived, or should one be governed by the element of time? The time element must be considered, because some cases will run their course and recover in from one to three weeks, even when untreated or badly treated. Also cases may recover after many weeks of purulent activity. Perhaps after three or four weeks of inflammation, which has not subsided, the non-acute stage has begun.

Referring to mastoid involvement, Dr. Meierhof said that in his judgment every infection of the middle ear—especially in infancy or early

childhood—involves the mastoid antrum. Many knew of cases where there were distinct evidences of mastoid involvement which had recovered, operation having been advised, but refused. However, cases are seen where there has been a pyogenic disease of the middle ear for many weeks or months, before coming under observation. What should be the course in these cases? If the patient has no urgent symptoms and has not been properly treated according to one's judgment, then the child should be given the benefit of such treatment as has yielded successful results. If the case shows no sign of improving within a few days, no time should be lost in opening the mastoid, whether there be symptoms of mastoid involvement or not. The time has passed when the advisability of opening the mastoid is dependent upon vital indications. The preservation of hearing alone is sufficient ground for arresting the destructive pyogenic process, especially so when due to violent types of infection occurring in the course of scarlet fever, diphtheria, pneumonia, and other infectious diseases. Even a staphylococcus infection in a non-resistant individual can be as destructive as the more virulent types in others.

In trained hands the danger from opening the mastoid is reduced almost to a minimum, but in spite of this we still have to encounter the fear and prejudice of an operation on the part of the family and, knowing this, we often postpone telling them what our real judgment is. Of course, where symptoms are urgent there is usually no difficulty in obtaining consent, but there are border-land cases that would do better if the mastoid were opened, even though recovery might take place without operation, whether in the acute or non-acute stage. It is just these cases which give rise to discussion, especially with private patients. In hospital practice one is more free to act according to his judgment. Then, what are the factors that give rise to an indication for a simple mastoidectomy in the non-acute stage of middle ear and mastoid infections? If evidences of mastoiditis are apparent, there is no need for further consideration—one should operate. If the case is one of long standing without mastoid symptoms, but shows no sign of yielding to approved treatment—with or without the presence of symptoms other than discharge—operation is also advisable.

Relation of Diarrhoea in Children to Otitis Media Purulenta. Dr. Herman L. Schwartz.

DISCUSSION.

DR. TRUMAN L. SAUNDERS said that Dr. Schwartz had so completely covered the subject that it only remained for him to emphasize the fact that there is a definite relationship between otitis and disorders of the digestive tract. This is brought out in clinical cases where the child suffers from disturbances of the digestive apparatus and the condition of the ear is not noticed until drum perforates and discharges and the child improves. We will, therefore, avoid mistakes and benefit our patients if in every case of digestive disturbance a routine examination of the ear is also made. In many of these cases if the ear is found to be involved, it will clear up by incision of the ear drum without any other special treatment.

DR. WILLIAM A. SCRUTTON said that it would seem more likely that the gastro-intestinal disturbance is due to the ingestion of a muco-purulent discharge originating in the posterior ethmoid region. This in all probability is also responsible for the purulent otitis.

DR. PHILLIPS said that the pediatricians do not always see these cases, but we get them in the great hospitals. Some time ago he had reported what he had thought was the youngest case ever operated upon for mastoiditis, a child four weeks old, with a post-auricular swelling which was opened and drained. He had reported this with considerable gusto, but his conceit had been taken out of him by the late Dr. Gruening, who mentioned a case only one or two weeks old.

DR. OPPENHEIMER said that some years ago he had occasion to operate on a patient of Dr. Schwartz's, who, at the age of three weeks, had a very definite mastoid condition.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

October 26, 1921.

The Optic Nerve in Sinus Disease. Dr. Colman W. Cutler.

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

Aeration of the Posterior Accessory Sinuses in Acute Optic Neuritis. Dr. L. E. White, Boston. (By Invitation.)

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

DISCUSSION.

DR. C. G. COAKLEY expressed his appreciation of the admirable papers just presented. Dr. Cutler's paper took up the subject from an earlier standpoint than in the cases of which Dr. White spoke. There seems little doubt that many of the acute nasal accessory sinus cases, if examined by Dr. Cutler's method, would be found to have a variation in the size of the blind spot, but the patients do not complain of that phase of it. On the other hand, in acute infectious sinus disease and chronic disease of the posterior sinuses, one is struck by the rarity of optic neuritis. In quite a large experience with sinus disease he could recall but one case of serious or any involvement of the optic nerve, when the sinuses contained pus. Therefore, the conclusion to be drawn was that the chronic suppurative and polypoid types of sinus—the kind to which Dr. White referred—do not result in optic nerve involvement very often. The cases that do come are the ones spoken of by Dr. White, and some of those spoken of by Dr. Cutler, with enlargement of the blind spot, beginning defective vision, or beginning optic neuritis.

Dr. Coakley agreed with Dr. White that the nasal examination in these cases is negative. The most careful examination of the mucous membrane fails to show any involvement, not merely the ordinary examination with the nasal speculum, but with the use of the pharyngoscope, which can be inserted after cocainization, rendering possible a careful inspection of the posterior portion of the nares. In that group of cases he has not found the least evidence of any congestion of the mucous membrane or any enlargement of the middle turbinate.

DR. WHITE had spoken as the probable cause of absence of recognizable pathological lesions being due to the absence of the aeration of the cells. In almost all of the cases that he himself had seen, and especially of the one to which Dr. White had referred, with the pharyngoscope one could see the orifice of the sphenoid and with a little care could pass a probe into it, and the palpation was practically normal, and when he had been urged to open the posterior and sphenoidal cavities there had been, as Wr. White said, an almost immediate improvement in vision; and if it did not improve within forty-eight hours, it was pretty evident that the sinus condition was not the cause of the condition. Sometimes he had been inclined to feel that this was due to the bleeding, which in some way or another had benefited the patient, but since all his operative work was done with cocaine and adrenalin, often the amount of blood lost was only two or three drops, so one could hardly account for it by the hemorrhage. One could see the membrane and it was almost white and with no polypoid degeneration . It was difficult to say what took place, but the patients got well. In some cases, where the ophthalmologist urged operation and where the conditions were practically negative and the patients refused operation, they have gotten well without it. The internists ascribe the result to the absorption of toxins, etc. How many of the cases that were operated upon and gotten well would have gotten well without it, it was difficult to say.

Referring to the X-ray examinations, Dr. Coakley said that in all the cases that he had seen, the X-ray plates had been perfectly normal and had been of no value. One side might be a little more clouded than another, but that might be due to some anatomical reason, or one cell

might not be quite so well developed. One must be very careful in taking the interpretation of the radiologist.

Dr. E. S. Thomson said that the interesting papers by Dr. Cutler and Dr. White opened up a large field for discussion upon a subject which has many new aspects. The role of purulent sinusitis in optic nerve disease had been much discussed of late, but he would confine himelf to lesion of the optic nerve, in which active sinus disease cannot be, or has not been, demonstrated.

Dr. Cutler's contribution upon the enlargement of the blind spot was of great interest. This is, as he said, an early symptom which is of less importance from the standpoint of prognosis than the failing visual acuity, central color scotoma, or haziness and redness of the optic disc; that is to say that the patients usually complain of failing central vision and color defects, and these are the most important guides for operation. Upon the constancy of the enlargement of the blind spot, as well as its value in differential diagnosis, he did not at the time wish to speak.

Dr. Cutler's classification of the cases into "acute" and "chronic" certainly brought out a very important clinical point. In the acute cases where the vision is failing rapidly, operation should be done as soon as the diagnosis has been made. In the chronic cases, the need is not so urgent, but is none the less important. In the class of cases under consideration, the diagnosis can often be made only by the eye symptoms. X-ray findings are negative, and when the sinuses are opened, they are not found to be manifestly diseased. The rapid recovery after operation, however, makes it positive that the sinuses have been at fault.

In the more chronic cases, the question of operation is more difficult, and probably depends on how deeply the bony disease has extended, if such exists, as he believes it does. In some cases of purulent sinusitis, the eye symptoms disappear if the sinus is thoroughly irrigated, and if the vision and color perception return to normal under these circumstances, it is certainly proper to wait. Failing this, or in the presence of the least tendency to whitening of the nerve, the sinuses should be opened without too long delay. There is no question but that in some cases the process is a limited one, and patients who refuse operation recover with a permanent scotoma or a greater or less degree of atrophy of the optic nerve. This atrophy is apt to progress later, and one must always bear in mind the delicacy and vulnerbility of the optic nerve fibres and not wait too long.

Dr. Thomson said he had in mind several such cases, in which no operation was done and in which the initial damage to the nerve was not great. Year by year the vision slowly fails and it was, in his experience, the great exception where such an unfortunate sequence did not occur. It is, therefore, highly important to save the optic nerve any damage whatever, if it be possible. A properly done operation on the ethmoids and sphenoid offers no great risk. There is certainly great danger in allowing a latent process to continue beyond the point of functional disturbance, and who shall say where this point may be.

Dr. Coffin said that the subject under discussion was full of interest from the etiological, pathological, diagnostic, and treatment standpoints.

Etiologically we are talking on the basis that the underlying cause of the optic nerve is the diseased sinus or sinuses. Two theories are advanced as to the manner in which the nerve is affected, and while thinking of the frontal nerve, one must not lose sight of the neighborhood changes in other tissues. These theories are: first, toxemia; second, pressure.

Toxemia is thought of on account of the intimate relationship of some of the accessory sinuses and the nerves. Pressure accounts better, probably, for all the phenomena connected with the case: for instance, the edema, often tortuous veins of the fundud, the sudden improvement following operative procedures or other such procedures as influence the local circulation for the better. Pressure is accounted for in two ways: first, by the swelling of the membrane lining the nerve canal; and, sec-

ond, by considering the nerve as lying within an edematous zone, whether this be in the nerve canal or elsewhere. The last is probably the proper explanation in the majority of cases. This pathologically serial picture would explain his position.

Summing up on etiology: Pressure from an edema due to thrombophlebitis is plausible for the following reasons: (1) It explains more of the phenomena present in most cases than does toxemia; (2) It is a condition that has been shown to exist; (3) the prompt recovery of the nerve after early operation; (4) the recovery of vision after suction and compression applied to the nose, which, in especially latent sinusitis, can affect only the circulation; (5) the administration of lemon juice—which has a tendency to reduce the coagulability of the blood—hastens recovery in these cases.

Dr. Coffin said, that in a paper read at Washington in 1910, in speaking of the difficulties of sinus disease diagnosis, he stated: "In many of these cases the ophthalmologist may help to a more positive diagnosis by a description of what he finds in the fundus of the eye and the study of the visual fields;" and in a paper read before the State Society in 1912, in speaking of latent occult sinusitis, he stated that "it may include any of the sinuses and is of the utmost importance because of the difficulty of diagnosis."

When confined to the ethmoids or the sphenoids, or the two combined, the diagnosis must rest upon symptoms and possibly upon the study of the eye. In the years that have intervened he has been more thoroughly convinced that many cases of accessory sinus disease can be positively diagnosed only by the findings of the eye. As for diagnosis then, more frequently should the patient be going from the ophthalmologist to the rhinologist with the diagnosis made and with a request for treatment.

The treatment may be operative along more conservative lines. Dr. Coffin said he had had some most gratifying results following the use of positive and negative pressure applied intranasally, and this in many instances when no pus could be demonstrated. His feeling has been that the beneficial results were entirely due to the resulting improved local circulation. He also felt that the administration of lemon juice in large quantities would often be an aid to quick recovery.

Dr. Harmon Smith said that, after such comprehensive papers as had been presented, it would be superfluous to dwell on the obvious, so he would say nothing about the suppurative conditions so well known to the ophthalmologists and rhinologists as one of the causes of neuritis. In the acute suppurative conditions where the X-ray bears out the other findings, every one, of course, knows what course to pursue, but those cases where the symptoms are remote and available methods are not definite in determining the cause of the neuritis are the ones which demand the most careful attention. Doctor White had not laid the emphasis on hyperplasia that he believed it deserved. Hyperplasia is probably the cause of more of the symptoms cited than any of the suppurative conditions.

Dr. Smith said that it was reasonable to suppose that in climates, such as this, that the hyperplasias existing in the turbinates of the majority of people could very well extend to the lining of the accessory nasal sinuses, and that in the cases examined for Doctor Sluder by Doctor Wright some years ago, they all presented varying degrees of hyperplasia, and while these examinations were made for the neuralgias of nasal origin, the same principle obtains in regard to the involvement of the optic nerve. The exception to this hyperplasia involvement is where there are some changes in the bony structure of the walls of the channels through which such nerves as the optic and the Vidian run. Of course, there might be a peri-osteitis or a rarifying osteitis, either of which would create pressure on the nerve from without inward, and in these cases no operative measures would overcome the condition. Hence our failure in some cases. Whether the neuritis is due to hyperplasia or other conditions already emphasized, the operation reduces

the pressure by depletion and aeration and the nerve usually regains its normal, if the proceedure is executed in time. In all cases where he had operated himself he had found a hyperplasia of the turbinated structure and no evidence of free pus in the sinuses themselves. The nearest to a purulent condition was a few granulations in one or two cases.

Doctor Smith did not believe that the diagnosis of these conditions should be thrown upon the shoulders of the rhinologist, as he had only transillumination, X-ray, suction, and pus to aid him in his diagnosis, and in nearly every instance one or more of these conditions are negative. Hence he believed that it is the function of the ophthalmologist to determine the necessity for operation, and to advise the rhinologist when to do it. Of course, this is the only means left after the exclusion of other causes of neuritis.

Dr. Ellice M. Alger said that the two extremely careful and interesting material had provided a great deal to think about. These sinus conditions are much more serious than is realized, for doubtless many of them get well spontaneously and never come under observation, but when they are seen they often cause no little perturbation. He thought he had often noticed a tendency to "pass the buck" between the rhinologist and the ophthalmologist, neither one being willing to assume the responsibility of ordering the sinuses opened. Dr. White had shown in his paper that the nasal and X-ray findings are very often negative and the decision to operate must be based largely on the ophthalmological findings. Dr. Cutler, on the other hand, had stated that even when there were definite evidences of retrobulbar neuritis, they might be due to several even more probable causes than sinus disease. Even enlargement of the blind spot, which is the symptom most suggestive of it, occurs in a number of relatively common conditions. However, if every patient examined as carefully as Dr. White's had been, and other lesions excluded as far as possible, opening of the sinuses was clearly called for. Unfortunately, many of these patients fall into the hands of men whose ambition and enthusiasm are out of all proportion to their diagnostic judgment and operative skill. All have seen the unfortunate results of these operations, and Dr. Cushing had recently called attention to the thoughtless way in which they are undertaken; the inexpert way in which they are often done, and the deplorable results. He has frequently seen meningitis after these supposedly safe proceeures.

So far as the ophthalmologist is concerned, the cases referred from the rhinologists are comparatively simple, because they have definite nasal symptoms, but the patients who came to him directly generally consulted him because of failing vision due to retrobulbar neuritis and not because their blind spots were enlarged. Few people have such perfectly healthy teeth, tonsils and digestion that the respective consultants would definitely exclude them without a considerable period of observation and treatment, and yet in the absence of evidence directly pointing to the sinuses, they must be excluded first.

Dr. Alger agreed with Dr. Cutler upon the ease with which the blind spot can be mapped out in most instances, and the great diagnostic value of its rapid enlargement. But, as in all subjective tests, much depends on the intelligence and the method employed. He had seen some patients in whom it was hardly possible to demonstrate the presence of any blind spot at all, while many others with apparently perfectly healthy eyes had spots larger than the average. He preferred the use of the stereoscope, with which it was possible not only to map out and compare rapidly the blind spots of both eyes, but to compare their central color sense, differences in which could be detected long before there was any definite scotomata for color.

Dr. F. M. Law said that, unfortunately, the X-ray does not give much evidence in these cases, but it is mainly the fault of the interpreter. The evidence is on the plate, but it is not recognized, and why? Simply because the rhinologists, the otologists and the ophthalmologists send their cases to the laboratory with merely a request for examination.

The radiologist does not like to make any more examinations than are necessary, and you get the report simply on a sinus plate; whereas, if the case were specified as coming from an ophthalmologist with a suspicion of an optic condition due to a sinusitis that would be met differently, in a special stereoscopic way, then there would be a better chance for definite information. We can get information from a plate which shows in a very small way. The surgeon cannot see it alone; the roentgenologist man cannot see it alone, but when they get together and discuss the case, these little points will come out and indicate the difference between a normal and a pathological condition. This depends on the clearness with which you can see the changes in the sphenoid and ethmoid walls. If there is a consultation we can learn what these bony changes mean, and therefore get more information from the X-ray plates. Be a little more careful in your requests for X-ray examination; specify the type of case; give the roentgenologist all the information possible, and then talk over the plates with him. By so doing, information will be obtained from the plates which otherwise would have been overlooked. The changes are very slight and easily misinterpreted.

Dr. Beaman Douglas said that it was impossible to discuss these important papers properly in the short time available, yet with such a large audience present there would doubtless be a large stimulation in the clinics and one important result would be accomplished; namely, that these cases would be more closely examined. As Dr. White had said, these conditions might be briefly grouped into toxaemias and pressure cases. Many of the toxic cases get well entirely by themselves, but the intestinal toxemias do not get well. The others that do not get well are due to pressure on the optic foramen. It was difficult to conceive of any ethmoidal or sphenoidal lesions causing a lesion in the optic nerve itself without pus and a tumor, but in the optic nerve along the canal where it is fixed in the canal, a little exudate, a little thickening of the tissues or increase of the blood supply would easily produce sufficient pressure to produce peripheral effects. There is an analogous situation in the antra and orbital nerve. We have there a foramen which is fixed and a nerve wall that is thinner than the antral wall; and there we have a pressure which is greater than the optic nerve and a cavity similar to the sphenoid, and yet we rarely have interference with the sensory nerve. Are not all these toxic, not pressure phenomena?

Again, the nose men must have a minimum basis on which to examine these cases and to decide. As Dr. Alger said, we are always "passing the buck." What are the minimal conditions? First, the middle turbinate body. It is not surprising that when cocain is used, these areas of hyperplasia and edema are not found, for, of course, they disappear under such conditions. Instead, the superior turbinate body should be examined with a probe; it is better than the X-ray, for it will discover little areas of congestion or hyperplasia, and rugosities will certainly be discovered: 1st, middle turbinate almost normal; 2nd, minimum rugosities; and 3rd, irritable sinus, as that cuts up as soon as the probe is passed into it, and gets up all kinds of redness and congestion.

Dr. Douglas said he had found this in a more limited number than Dr. White had met with, but it had been most useful in his hands. He congratulated Dr. White upon the work he had accomplished, and said that it had been much appreciated.

Dr. T. Passmore Berens thanked the readers of the papers for calling attention in so masterly a manner to this serious condition, so generally overlooked by the profession at large. He said further: "The purulent cases are easily handled. The diagnosis is readily made, not infrequently by the rhinologist; at least sufficient suspicion is aroused to cause him to refer the case to the ophthalmologist.

"The cases to which Dr. White has called attention are not by any means so simple. In these, we must depend on the ophthalmologist for diagnosis. In the cases of rapid development, it is better to perform at least a conservative operation at once, and to theorize later.

"The cause of the hyperplasia is a long way from being cleared up. Personally, I believe there are many contributing factors. One of the most frequent is a digestive disturbance of some sort—whether from the teeth or tonsils—or most probable of all, a straight intestinal toxemia. Frequently intestinal toxemias will result in recurrent congestion of the mucous membranes of the nose and of the accessory sinuses, more especially in the case of deformities of the bony framework and of the turbinals, and it seems to me that such cases are apt to get well without operation."

DR. J. L. HARRIS expressed much gratitude to the essayists of the evening. Dr. Cutler's paper was one of the most scholarly that he had heard for a long time, and as for Dr. White's work, he had been carrying on a campaign in the face of no little criticism at times until he had brought it to the attention of the profession in a meeting which was phenomenal in the history of the section, and he deserved very hearty congratulations.

Dr. Harris said he would like to emphasize one point among the many that had been referred to—the economic feature of this subject. It had been his privilege last winter to hear Dr. White present a paper which had impressed him very much, and at that time he showed a young lady, 17 years of age, totally blind. She was helped in and placed in a chair. Dr. White said she was a hopeless case, and after she went out, said that in his judgment, it was all a matter of neglected surgery, and that when he got hold of the case it was too late. It would be a great pity if any went away from this meeting befogged, as some are apt to do. Dr. Harris said that in his judgment the ophthalmologists and laryngologists would leave the meeting so clear in diagnosis, and on the points that Drs. White and Cutler had made, that in future they would, none of them, "pass the buck," but would establish a diagnosis based on the symptoms; and if they have in a patient with rapidly waning vision, an increase of the blind spot, but one thing would be present, namely, disease of the nasal sinuses. Dr. White had pointed out in a long series of cases as Dr. St. Clair Thompson had also done, that the work could be done in a very simple way, often by turbinal surgery, with no need of clearing out the ethmoid cells, with a very happy effect.

Dr. MacPherson asked what do we mean by a toxemia? Is it a toxemia carried around from the sinuses through the general circulation, passing through the heart and the lungs and lodging in the nerve, or is it a toxemia passing from structure to structure and attacking the nerve as a result of anatomical relationship?

Where the papillitis is due to the former, we should find it accompanying pus focci, occurring in the tonsils, teeth, gall bladder, etc. Moreover, where one eye only is involved, treatment is directed, as a matter of course, toward the sinus of that side and not to the opposite, and relief is obtained, indicating that the toxic substance, if such it is, is not usually transmitted through the general circulation.

Suction where pus is present, or where there are no pus signs apart from ordinary hypertrophy, is an aid to diagnosis. Inasmuch as the nerve oedema frequently subsides under such treatment, and thereby indicates the nose to be the seat of the disturbance.

DR. M. COHEN said that Van der Hoer's work on the blind spot, published some years ago, prompted him to examine several hundred cases suffering from atrophic and hypertrophic rhinitis. He could not convince himself that the blind spot was enlarged in the majority of the cases examined in the clinic.

DR. MACKENTY said he did not feel competent to discuss Dr. Cutler's paper, but Dr. White's paper had interested him very much, and felt that his work in this particular field was a classic.

He had evidently spent much time and work in painstaking investigation in order to produce such a paper. It was all scientific work.

Dr. MacKenty said that while traveling this summer, he consulted many men on this subject. He had taken interest in finding out the feeling of men in other parts in regard to this particular subject, and brought out the opinions that a purulent disease must be present in all cases. They could not see sinusitis without pus.

There is no question but that here we are facing another pathology which is not the same that produces pus. He did not like the term "negative findings." There are always findings if you look carefully and often enough for them. A careful history of the case is essential and generally points to the sinuses. The local examination, if properly and carefully done, will give nasal findings. Of course, if the nose is adrenalized and cocainized before it is inspected, nothing may be found. Dr. Douglass said very truly, that hyperplasia is the first pathological factor; other factors come secondarily. What causes hyperplasia? That is what we want to know. Dr. MacKenty said he thought that suction in these cases is a willful waste of time.

Dr. MacKenty said that Dr. White's paper presented one of the most lucid pathological definitions of the condition that we have had. It covers the point up to where we have arrived in our clinical observations. He did not, however, quite agree with Dr. White's conservative operation and himself preferred a more radical one. Neither did he think Dr. White's operation simple. A more radical procedure seemed to him easier of accomplishment and he was for early and radical operation in all cases, especially in the acute ones.

After all, however, the rhinologist has generally to operate on the opinion and findings of the ophthalmologist, since his advice and direction are of supreme importance on whether an operation should be done or expectant treatment adopted.

Dr. Voislawsky said that everything he might have said had been already covered by the rhinologists.

Dr. Conrad Berens, Jr., emphasized two points: (1) Infection at the apices of the teeth may produce enlargement of the blind spots, which should be ruled out before radical measures are undertaken, although it occurred only once in the examination of 1700 patients. This case, M. P., 52 years of age, a white man, was first seen on October 17, 1916. The corrected vision was: R.E., 20/30; L.E., 20/15. Fundi, normal. Right blind spot, irregularly enlarged; left, normal. General physical examination and examination of the sinuses were negative except for three abscesses at the apices of devitalized teeth. These teeth were extracted, and on November 9, 1916, the vision was: R.E., 20/20; L.E., 20/15. Blind spots, normal.

(2) Early recognition of optic nerve involvement by the rhinologist.

Two signs which are easily elicited, added to the well-known symptoms of blurring of vision and subjective scotomata in the visual field, should lead to careful study. The first is retro-bulbar tenderness, and the second is the confrontation test for the determination of the size of the blind spot. The confrontation test, as usually described and practiced, has proven untenable—at least in his hands—due to the examiners' inability to maintain a fixed distance with the test object; however, with the aid of a modified Maddox rule, or with an ordinary string one meter in length, the test may be made with accuracy. The end of the meter string is held by the patient on a level with the patient's right eye, and the observer holds the other end on a level with his left eye. The eye not under examination is closed or covered, and a white headed pin passed through the string at 50 centimeters is carried temporally slightly below the horizontal, eye fixing eye, until the blind area is found and compared with the observer's. This method is rapid but accurate, for it controls distance and fixation, and with a little practice will be found an easy clinical test for the ophthalmologist as well as for the rhinologist.

Dr. S. McCullagh called attention to the fact that in his experience this disease was a disease of early life—often of childhood. In the

past year he had seen seven cases of retrobulbar optic neuritis, four due to sinus disease of the hyperplastic type. Three of these were in children under 14 years of age, and one was a girl of 18. The other three cases were due to the purulent type, all these patients being under twenty-five years of age—early adult life. He asked if that corresponded with the observation of the other generation, especially as to cases due to the hyperplastic type of sinus disease.

Dr. Cutler, in closing, thanked the audience for their courtesy in listening to a paper on an alien subject. The principal object in presenting the paper was to call attention to the subject of chronic cases. The acute cases were in the hands of the rhinologists and were self evident, as a rule; but the chronic cases call for the utmost care in diagnosis and the elimination of many things. The blind spot is not always varied enough to give definite signs. The symptoms ought to be studied by a good many men before final and conclusive data can be obtained.

Dr. Cutler said that some who had spoken on the subject were inclined to think that the central vision is generally affected first, for the patients naturally complain of what is most obvious; but in the cases of recurrent colds and catarrhal conditions, and especially with tenderness on pressure of the eyeballs, the blind spot ought to be examined, and the problem is then what to do with the chronic cases. It would be interesting to have an evening devoted to that particular point. We ought to examine many more cases than we do and examine for the blind spot, even if the patient does not report a blind spot of vision.

Dr. L. E. White (Boston) in closing, called attention to the fact that cases recover after various methods of operating and under various forms of treatment. He said he felt as Dr. MacKenty did, that if the nasal condition is responsible for the infection or swelling about the optic nerve, the nose will not be negative to the careful examiner. Many rhinologists do have enough cases to comprehend what they see when looking at these noses. If the middle turbinate is acutely swollen and not chronically enlarged, its removal is not advocated. He thinks it is extremely important to differentiate between the blocking by the acutely swollen middle turbinates where ventilation can be obtained by the simple process of shrinking, and those in which this ventilation cannot be obtained because the turbinate has become chronically hypertrophied. He cited a case of a patient without even light perception, who improved so much after cocainization and hot irrigation that fingers could be distinguished at several feet. After one or more similar experiences he felt that by waiting a day or two many cases might recover without operation. On the other hand, he cautioned against needless delay, as optic atrophy may occur while waiting for recovery. It was to illustrate this point that he showed the case mentioned by Dr. Harris. This young girl, 4 years ago, had a sudden blurring of vision. She was early examined by an eye man, who found a well developed case of unilateral optic neuritis, and referred her to one of our best hospitals for treatment. When she entered this institution 12 days after the onset of the neuritis, the vision had dropped to light perception. A very thorough and complete examination was made, but no neurological condition was found to explain the loss of vision. As the patient did not complain of nasal obstruction and the roentgenograms were negative, nothing was attempted in a surgical way, and she was discharged unimproved at the end of two weeks. The nerve in this eye went on to complete atrophy. Nine months later the other eye was similarly affected and she was treated as a house patient at another institution. Again no attempt was made to relieve the pressure about the optic nerve and complete atrophy followed, so that when Dr. White showed her 4 years later she was completely blind. This girl had marked obstruction to the ventilation of her posterior sinuses and an early operation, in all probability, would have saved her vision. It is only when one sees these pathetic cases which might have been helped that one realizes the responsibility of acting

intelligently before it is too late. Dr. White said that this case, above all others, had stimulated him in his efforts to impress upon the ophthalmologists and rhinologists the necessity for a correct diagnosis and prompt operative interference. While these bad cases are rare, they do occur and unless one is awake to their seriousness, permanent blindness, as illustrated by this case, may follow. Even although a case does recover under local treatment, there is danger of recurrence, so that if the condition in the nose predisposes to recurrence an operation to permanently ventilate the accessory sinuses would seem to be indicated. The operation required is simple and almost devoid of danger, not for a moment to be compared with the trials that this girl, for instance, must endure all her life. Dr. White said that Dr. Cushing had emphasized the fatalities in these cases, but these had resulted, as far as he was able to determine, from unnecessarily extensive operative procedures and on cases where the loss of vision was usually due to intracranial conditions and not to nasal infection. While it is important that the dangers of unnecessary operation should be emphasized and greater care taken in making a differential diagnosis, it is, on the other hand, of the utmost importance for the ophthalmologists and rhinologists to remember that untreated optic neuritis frequently leads to permanent blindness. Dr. Cushing, at a meeting of the New England Otological and Laryngological Society a year ago, had shown a case dying from meningitis, the result of unnecessary nasal operating. In this case (a man with acoustic tumor) the intracranial pressure had so thinned the bones adjacent to the accessory sinuses that the dura was forced down into the nose (which had been weakened by several intranasal operations) as was shown at autopsy. Dr. White does not think there is any excuse for these nasal operations on patients with brain tumors. As scientific men, it is of the utmost importance that careful study be made of each case. Just because a patient with loss of vision has nasal pathology, it does not necessarily follow that the nasal condition is responsible for the loss. In every case intracranial tumors must be considered, hence the necessity for the visual fields, the study of the fundus, the history of the case and a very careful neurological examination. While these examinations take time and patience, they make for accuracy in diagnosis. To operate needlessly, especially with resulting fatalities, is even worse than permitting one to go blind through neglect; but with intelligent study of the cases, both extremes should be avoided.

In referring to the negative findings mentioned by Dr. Coakley, Dr. White said that whenever it is possible to see the opening to the sphenoid with a naso-pharyngoscope the case would probably recover under treatment. He also said that while he had not made much use of the suction treatment mentioned by Dr. Coffin, he felt that the recoveries following its use would probably have taken place without it. He did not feel that too much reliance should be placed upon it in serious cases; cases, for instance, where there might be an exudate about the nerve where needless delay should not be encountered. Many of the cases he had treated were of a very serious nature, having, in several instances, been treated elsewhere for a considerable period and it was urgent that the pressure on the nerve be relieved at once. In the milder cases, where the necessity of prompt relief was not urgent, there was no objection to the use of the suction method. He mentioned that he sees mild cases recovering within a week or ten days without any treatment.

Dr. Thomson had expressed well the views he held in regard to recurrences. In every case there is a chance of recurrence, so if there is any condition in the nose suggesting this danger, the case should be operated upon, even though the patient is in a satisfactory condition at the time. Dr. White mentioned he had recently operated on a patient with post-neuritic atrophy. He did this because of the history of exophthalmos and found the posterior ethmoid cell filled with polypoid tissue and felt it was worth a great deal to the patient to have this removed, as its presence would tend to recurrence. A reinfection in this region

might not only have produced an orbital abscess, but also the possibility of a cavernous sinus thrombosis. While this patient's vision will probably not be benefited, the danger of further loss is safeguarded.

Dr. Coffin had spoken of blood letting versus aeration. It is possible that the depletion incident to any operation on the nose benefits these cases, but where an enlarged turbinate causes blocking, its removal is indicated, not only to relieve the present condition, but to safeguard the patient's future. Blood letting where there is not marked blocking and where a more extensive operation is not indicated, might be of considerable immediate benefit.

As Dr. Douglas had said, the probe is a very valuable aid in determining the size and condition of the middle turbinate and its use should be encouraged. With it one can obtain the evidence that cannot be gained with any other method. Dr. Berens had spoken of not operating unless there was evident obstruction and felt that such cases would probably recover without operation. Dr. White said that he had practically arrived at this conclusion; that he was following these cases very closely, trying to decide which would recover with, and which without, operation.

Dr. Alger had spoken of not operating until after a very thorough study of the case, a point that Dr. White felt had been well covered in the paper. One should have not only the co-operation of the ophthalmologist, but also the internist, the neurologist and cerebral surgeon.

Referring to what Dr. Law had said about radiographs, Dr. White said that only stereoscopic plates were of much value. In some instances indistinctness of the ethmoid partitions had been noted, but in others not found. While much had been hoped from carefully timed radiographs, they had been rather disappointing as aids in diagnosis, negative plates by no means indicating that infection was not taking place. He advocated, as Dr. Law suggested, that the radiologist, the ophthalmologist and rhinologist confer as to the conditions and together study these plates. Radiograms are helpful in determining the size of the accessory sinuses and the depth of the sphenoid and are frequently consulted when operating. The radiograms were negative in the case to which he referred where a large amount of soft tissue was discovered in the posterior ethmoid. Dr. White mentioned a case of retrobulbar neuritis due to toxemia from diseased teeth. The nose was negative and he let it alone, but had the teeth extracted. Another case came from the tonsils and pus was evacuated on their removal. Both cases made uninterrupted recovery. Two cases had antrum disease and washing out the antra was of distinct advantage. One should be on the lookout for any infective process and eliminate it wherever found.

Dr. MacKenty had expressed this point very well. Dr. White said he was much interested as to the views of the men interviewed by Dr. MacKenty in Montreal as to the necessity of purulent infection. He had always had these views to contend with and they were still prevalent. Dr. White said that he saw so many of these cases without pus that he felt it would be better to look with suspicion upon it when found, than to longer consider it the only cause of optic neuritis. When pus was found in the nose the pressure in the tissues and sinuses was being relieved and the case was probably not as serious as those where it was not found. Dr. MacKenty had spoken of opening the sphenoid through the ethmoid labyrinth. Dr. White said that it was largely a matter of the personal equasion, one operator being trained to reach this cavity one way, and another, some other way. Personally he felt that the ethmoid exenteration was unnecessary and that a simpler operation answered all purposes, especially as Dr. Cushing had mentioned that he had seen several cases of meningitis following ethmoid exenteration.